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MANORANJAN MITRA
1895–1942

MANORANJAN MITRA
1895–1942

B. B. Mundkur

Manoranjan Mitra, Assistant Mycologist at the Imperial Agricultural Research Institute, New Delhi, since 1921, died on the 10th of July, 1942, at his home at Hazaribagh, following diabetic coma.

Mitra was born at Amritsar on June 19, 1895, third son of a gifted family of Bengali Christians, who had settled down in the Punjab. During his boyhood he attended the Central Model School at Lahore from which he was graduated in 1912. He entered the Punjab University (Government College), Lahore, the same year and received the B.Sc. degree in 1916 and the M.Sc. degree in 1918, majoring in botany, first class, first on the roll. His M.Sc. thesis was on the anatomy and development of some Himalayan species of *Adiantum* and *Pteris*.

He served for a time as a lecturer at St. John's College, Agra, but his interests lay, however, in research and in 1919 he came to the Institute as a graduate student to get training in mycology under Sir Edwin J. Butler F.R.S. who was then the Imperial Mycologist. In 1921 the Associateship of this Institute was conferred on Mitra. As a graduate student he worked on an *Acrothecium* attacking bajra (*Pennisetum typhoides*), to accommodate which he established the species *Acrothecium penniseti*.

Soon after the completion of his training, Mitra was appointed Assistant Mycologist, a post that he held until his death.

In 1926 he was deputed by the Government of India to survey the crop diseases occurring in the Andaman Islands and in 1927 he was granted sabbatical leave to proceed to England. He joined the Imperial College of Science, London University, where his investigations were mainly on the species of *Helminthosporium* affecting Indian cereals. He was granted the diploma of the Imperial College in 1929 and the Ph.D. degree of the London University the same year, and that of D.Sc., in 1936.

Mitra's chief interest lay in the diseases of cereals, especially those caused by species of *Helminthosporium*. The earlier accounts of this genus failed to distinguish the Indian species correctly but as a result of Mitra's investigations, our knowledge of this genus is now on a stable basis. In 1930 he discovered a bunt of wheat in the plains of India, now known as the "Karnal Bunt," and named the fungus *Tilletia indica*. He has two more species to his credit, *Helminthosporium bicolor* and *Helminthosporium frumentacei* and a variety, *Helminthosporium halodes* var. *tritici*.

Mitra was a man of retiring nature; he felt more at home among his fungi and his books than elsewhere. His knowledge of Indian plant diseases was very profound; and the graduate students of the mycology section found in him a scholarly teacher. He was a member of the Linnean Society, British Mycological Society, The American Phytopathological Society and

the Indian Botanical Society. He leaves his wife and two sons and a large circle of friends to mourn his loss.

Mycology Section,
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2. Morphology and parasitism of *Acrothecium Penniseti* n. sp. (a new disease of *Pennisetum typhoideum*). Mem. Dep. Agr. India, Bot. Ser. **11**: 57–74. 1921.
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STREAK AND MOSAIC OF CINERARIA[1]

LEON K. JONES

(Accepted for publication April 25, 1944)

INTRODUCTION

During the winter of 1937–38, a disease of cineraria was noted in one greenhouse in Spokane, Washington. Necrosis of leaves and stems often developed to such an extent that most of the leaves on affected plants died progressively up the stem and many plants wilted and died previous to, and during, blossoming. The disease was called streak[2] and was investigated in the Experiment Station greenhouses at Pullman, Washington, and in commercial greenhouses in various localities in the State to determine its nature and the methods of control. A disease of cinerarias with symptoms similar to those associated with streak was reported in 1934[3] as caused by the spotted wilt virus. The present studies included tests to determine the possible relationships of the 2 diseases.

In a greenhouse in Walla Walla, Washington, in January, 1938, 2 per cent of the cineraria plants were affected with a typical mosaic disease. The leaves were mottled with large, raised, dark green areas in the otherwise light green tissue and were puckered and malformed, and the plants were somewhat dwarfed. In the literature one reference to a mosaic disease of cineraria was noted.[4] The briefness of this reference makes it impossible to determine the exact nature of the mosaic-like trouble or its possible relationship to the mosaic disease observed in Washington. Lettuce mosaic has been transmitted to groundsel (*Senecio vulgaris* L.).[5] Groundsel is very closely related to cineraria and cinerarias are listed by some taxonomists as a development out of *Senecio cruentus* D. C. Other taxonomists prefer to classify cinerarias under the binomial, *Cineraria cruenta* Mass. Since lettuce mosaic produces a mild mottle on groundsel, these investigations included tests to determine if cineraria mosaic was caused by the lettuce mosaic virus which is commonly transmitted in lettuce seed. Further studies were made to determine the nature of the cineraria mosaic virus and measures of value in reducing losses from this disease.

SYMPTOMS

Streak

In commercial stock, the disease ordinarily does not become destructive until the plants approach blossoming, although the ruffling and rugosity of

[1] Published as scientific paper No. 596, College of Agriculture and Agricultural Experiment Stations, State College of Washington.

[2] Washington Agricultural Experiment Station Annual Reports **49**: 61. 1939; **50**: 77. 1940; **51**: 84. 1941; and **52**: 77–78. 1942.

[3] Gardner, M. W., and O. C. Whipple. Spotted wilt of tomatoes and its transmission by thrips. (Abstr.). Phytopath. **24**: 1136. 1934.

[4] Dickson, B. T. A mosaic-like disease of Cineraria. Ann. Report Quebec Soc. Prot. of Plants **1920**: 46–47. 1920.

[5] Ainsworth, G. C., and L. Ogilvie. Lettuce mosaic. Ann. Appl. Biol. **26**: 279–297. 1939.

leaves of younger plants indicate infection (Fig. 1, A). Rugosity and curling of leaves of young plants are very evident as the first symptoms following artificial inoculation. Reddish-brown areas in leaf veins (Fig. 2, C), petioles, and stalks of middle-aged plants may be observed with strong light transmitted through those areas. These areas may enlarge and constrict the affected tissues, which leads to necrosis of large triangular areas in the leaves (Fig. 3, B), necrosis of entire leaves (Fig. 4), or death of the plant. Wilting, rolling, yellowing, and death of leaves (Fig. 4) are commonly noted as the affected plants approach blossoming. Under some conditions, numerous, small, brown necrotic spots in the leaves are associated with the trouble

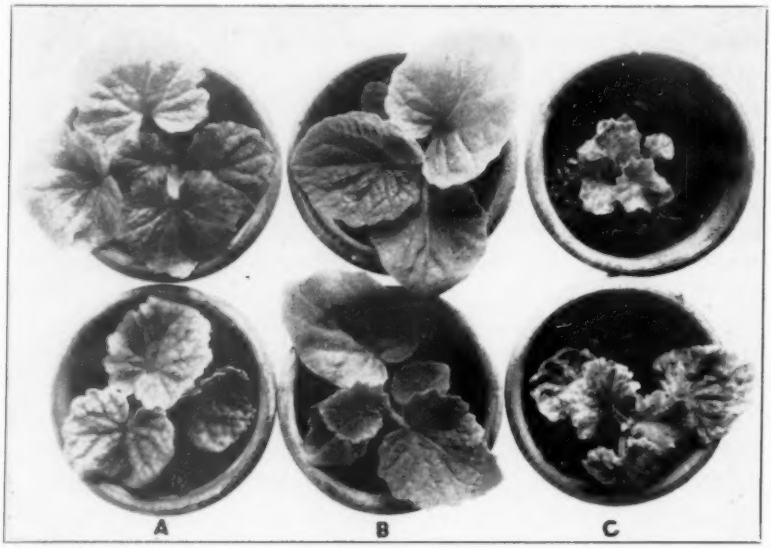

FIG. 1. Cineraria seedlings showing symptoms of streak (A), and streak and mosaic (C), compared with healthy seedlings (B). The plants became infected with the viruses by seed transmission.

(Fig. 3, A). The necrosis often develops to such an extent that most of the leaves die progressively up the stem and very few leaves are left on the plant at blossoming time. Affected plants wilt more easily than healthy plants and do not recover from wilting as readily.

Mosaic

The disease shows as mottling of the foliage with irregular light and dark green areas (Fig. 2, A and B). Crinkling, ruffling and dwarfing of leaves (Fig. 2, A), as well as shortening of leaf petioles and dwarfing of plants, are symptoms commonly associated with mosaic. Vein-clearing and puckering of young leaves following inoculation by mechanical methods precedes the appearance of the typical mottling.

Streak and Mosaic

The 2 diseases are often noted in the same plant, and a combination of

symptoms appears (Fig. 1, C). Plants are more severely dwarfed than are plants affected with either disease alone. Necrosis often appears in the light areas of the mosaic pattern in the middle-aged leaves before yellowing and necrosis appear in the older leaves.

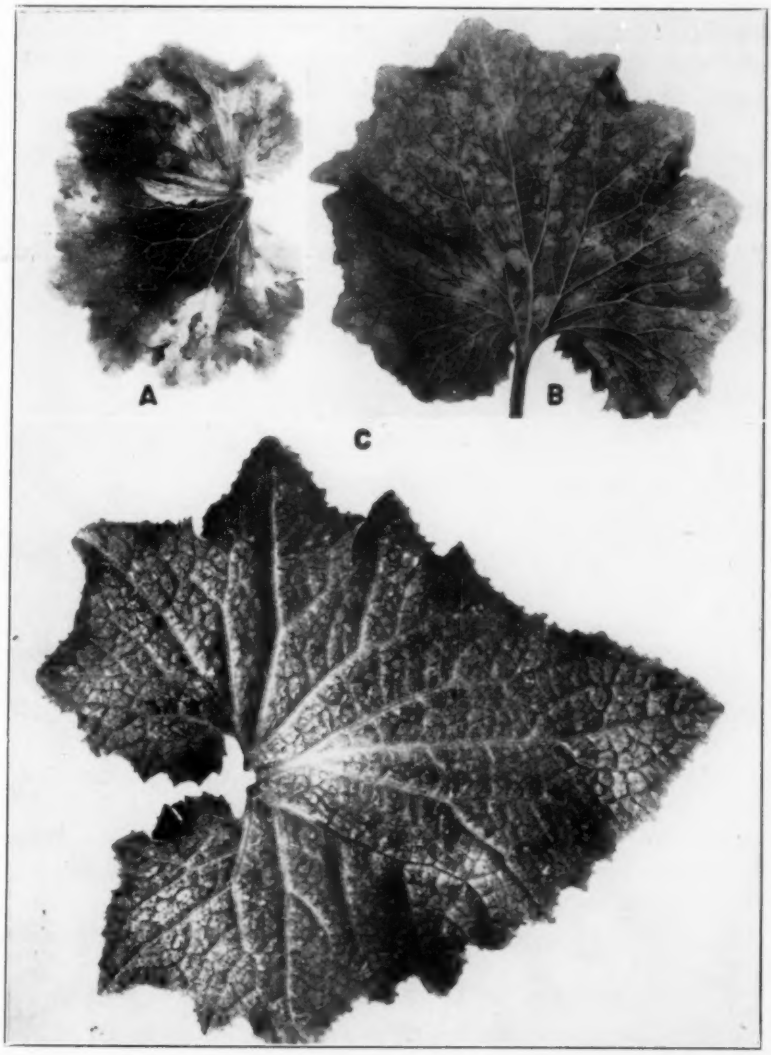

FIG. 2. Leaves of cineraria with mosaic and streak symptoms. A, ruming, rugosity, and mottling on young leaf, and B, mild mottling of older leaf caused by the mosaic virus. C, necrosis of leaf vein caused by the streak virus.

ECONOMIC IMPORTANCE

Streak

In the greenhouse where streak was first noted, 1200 to 1500 cinerarias are grown each season. The grower estimated that one-half of his plants in

1936, and possibly one-third in 1937 and in 1938, were discarded because of the presence of streak. Many diseased cinerarias were exhibited by growers in plant clinics held in Seattle, Washington, in 1939, and Spokane, Washington, in 1940, at conventions of the Northwest Florists, and the opinion was expressed that it was a serious problem that had developed only during the preceding few years.

Observations in greenhouses in Washington from 1938 through 1942 have shown that streak was usually present in the cineraria crop with the exception of crops produced by a few growers who saved seed from their own plant selections (Table 1). Counts were made to determine the prevalence

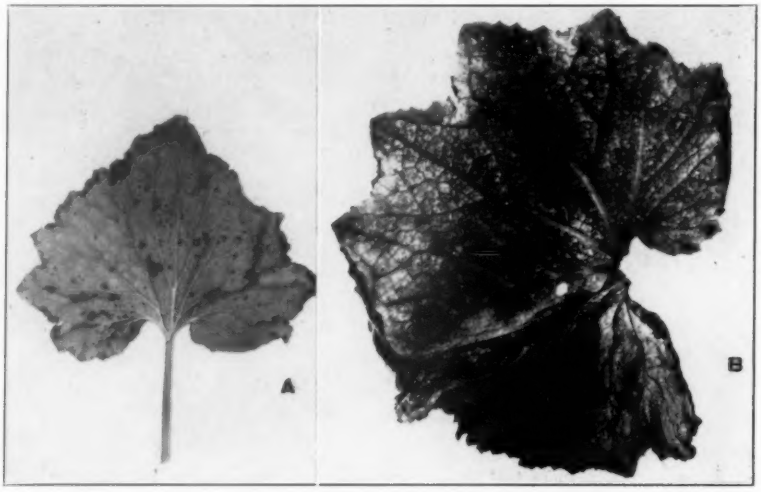

FIG. 3. Necrosis of cineraria leaves caused by the streak virus. A, scattered, brown necrotic spots and B, large necrotic area following vein necrosis.

of streak in the stock, but it was not possible to obtain data on the losses caused by the disease beyond determining the percentage of plants affected. Probably a high percentage of the affected plants was unsalable. The sale of affected plants also leads to an unfavorable reaction by the buying public, since such plants ordinarily have very poor keeping qualities when transferred from the humid greenhouses to the relatively dry atmosphere of residences.

Mosaic

In a greenhouse in Walla Walla, Washington, in January, 1938, about 2 per cent of the cineraria plants observed were affected with mosaic. The disease is not very prevalent, but is quite generally distributed (Table 1) since it was observed in Spokane, Walla Walla, Seattle, and Kent, Washington. Severely affected plants may be sufficiently dwarfed to be unsalable but, in general, the disease does not materially affect the salability of plants.

ETIOLOGY

The mottling-type of disease appeared very definitely to be a typical

mosaic caused by a virus. The streak disease, however, showed symptoms that are often associated with disease caused by parasitic organisms. Forty-four attempts to isolate an organism from necrotic areas in petioles, stems, and leaves of streak-affected plants were made in January and February of 1938. No fungi appeared on agar plates and bacteria occurred in only 3 instances. The failure to isolate an organism consistently from diseased tissue indicated that a virus was probably responsible for the trouble.

FIG. 4. Rolling, wilting, and death of cineraria leaves following inoculation with the streak virus.

Inoculations, by mechanical transfer of the virus, were made from diseased plants to healthy plants to determine if viruses were responsible for mosaic and streak. In 3 tests, 40, 50 and 82 per cent of the mosaic inoculations were successful, with an incubation period of 20 to 24 days. Sixteen, 50 and 50 per cent of the streak inoculations were successful, with an incubation period of 14 to 35 days. Since these preliminary tests showed that both streak and mosaic were caused by viruses, detailed studies were made to determine the characteristics of the viruses.

SEED TRANSMISSION

Some strains of cineraria stock in greenhouses in Spokane, Walla Walla, and Seattle in 1938 and 1939 were severely affected with streak or mosaic,

TABLE 1.—*The prevalence of streak and mosaic in cineraria plants in greenhouses of Washington*

Greenhouse	Year observed	Source of seed[a]	No. plants grown	Percentage[b] of plants with symptoms of	
				Streak	Mosaic
1	1938–1939	Local	1000	50	0
1	" "	Commercial	500	10	0
2	" "	"	800	0	5
3	" "	Local	800	0	0
4	" "	"	1000	20	2
5	" "	"	1500	0	0
5	" "	Commercial	400	50	10
6	" "	"	900	1	0
7	" "	"	600	50	0
8	" "	Local	1600	0	0
2	1939–1940	Commercial	800	20	0
9	" "	"	1200	20	0
1	" "	Local	1000	40	0
10	" "	"	1200	20	0
1	1940–1941	"	1200	25	0
1	" "	Select	500	0	0
10	" "	Local	800	75	0
11	" "	Commercial	300	50	0
12	" "	"	650	50	0
13	" "	"	900	75	0
14	" "	"	500	50	0
6	" "	"	1200	5	2
15	1941–1942	Local	800	0	0
15	" "	Commercial	1000	40	0
16	" "	"	300	20	0
8	" "	Local	1000	0	0
8	" "	Select	500	0	0
1	" "	Local	1200	10	0
1	" "	Select	600	0	0
5	" "	"	1500	0	0
10	" "	"	800	0	0
10	" "	Commercial	500	15	1
9	" "	"	500	20	2
9	" "	Select	600	0	0
17	" "	Commercial	950	75	0
18	1942–1943	Select	100	0	0
8	" "	"	800	0	0
10	" "	"	500	0	0
7	" "	"	500	48	0
17	" "	"	600	65	0
17	" "	Commercial	400	68	0
1	" "	Local	1000	52	0

[a] Local—seed collected by local growers; Commercial—seed purchased from commercial seed companies; Select—seed selected from virus-free plants at the State College of Washington.

[b] Percentages—based on random counts of at least two 100-plant lots in each crop observed during the months of January to March.

while other strains in the same greenhouse were free of these diseases. A strain of cineraria developed by one grower had considerable streak each season, not only on plants grown by the originator, but also on plants grown

from the same seed in 2 other greenhouses. Three strains of cineraria grown by local growers from seed of their own selection were free of streak in 1938–1939, although one strain had a low percentage of mosaic. Stock grown in a number of greenhouses from seed purchased from various seedsmen (Table 1) usually had varying percentages of streak, and stock from 5 of 16 commercial-seed strains had low percentages of mosaic. These observations indicated that each virus might be transmitted in the seed. Accordingly, several samples of seed were obtained from leading seed companies and local growers for virus-transmission tests in the greenhouses at Pullman, Washington (Table 2). Seed was also collected from mosaic- or streak-affected plants and tested to determine if the viruses were carried in the seed (Table 3).

TABLE 2.—*The transmission of mosaic and streak viruses in commercial seed. Pullman, Washington. 1938–1942*

Lot, year and variety of seed tested	Source of seed	No. plants grown	Percentage of plants with symptoms of		
			Mosaic	Streak	Mosaic and streak
1938–1939					
1 Grandiflora Nana	Seedsman No. 1	178	24	69	7
2 Howard & Smith	" " "	44	70	0	0
3 Prize mixed	" " "	129	5	20	2
4 Prize	Seedsman No. 2	130	9	32	5
5 Siter's Rainbow	Seedsman No. 3	70	0	57	0
6 Multiflora Nana	" " "	270	0	26	0
7 Hybrida Grandiflora	Local grower	53	0	47	0
1939–1940					
3 Prize mixed	Seedsman No. 1	36	0	42	0
4 Prize	Seedsman No. 2	139	0	35	0
5 Siter's Rainbow	Seedsman No. 3	146	0	34	0
7 Hybrida Grandiflora	Local grower	100	0	30	0
1941–1942					
8 Improved Prize	Seedsman No. 2	234	0	10	0
9 Prize, reselected	Seedsman No. 4	235	0	17	0
10 Hybrida mixed	" " "	117	0	14	0
11 Hybrida Grandiflora	" " "	207	0	20	0
7 Hybrida Grandiflora	Local grower	234	0	20	0

Each lot of seed was planted separately in flats and handled independently during potting. The hands were washed thoroughly with soap and water previous to handling each lot of plants. Where sufficient seed was available, a portion was used in 1938 and the remainder in 1939, except that the seed in lot 7 from a local grower was used again in 1941–1942 (Table 2).

The seed was planted in flats during August each season. The young plants were placed in 2½-inch pots in September and repotted to 4-inch pots in December. Plants with definite mottle symptoms of mosaic, or necrosis of streak, were discarded from each lot as soon as observed. Aphids were controlled as soon as observed by fumigation with nico-fume powder, but no attempt was made to control thrips. Accordingly, the percentages of plants listed in Tables 2 and 3 as showing symptoms of mosaic probably give a true

indication of the seed transmission of this virus. The percentages of plants with symptoms of streak, however, probably include considerable current season infection from a lower percentage of plants originally affected from seed transmission of the virus. Ruffling and rugosity of the foliage of young plants affected with streak indicates the presence of this virus, but this symptom alone was not considered sufficiently definite to discard plants. The

TABLE 3.—*The transmission of mosaic and streak in locally selected seed. Pullman, Washington. 1940–1942*

Plants from which seed was selected and year tested	Diseased condition of seed plant[a]	No. plants grown	Percentage of plants with symptoms of	
			Mosaic	Streak
1940–1941				
1	Streak	212	0	64
2	"	95	0	91
3	"	37	0	92
4	"	34	0	94
5	"	82	0	83
6	"	50	0	94
7	"	98	0	88
1–7 mixed	"	110	0	96
8	Mosaic and streak	240	20	79
9–13 mixed	Virus-free	49	0	0
17	Mosaic	250	19	0
18	Mosaic and streak	83	35	27
19	" " "	50	24	10
20	" " "	61	100	50
9	Virus-free	200	0	0
10	" "	144	0	0
11	" "	156	0	0
12	" "	120	0	0
13	" "	42	0	0
14	" "	46	0	0
14–16 mixed	" "	220	0	0
1941–1942				
1	Virus-free	180	0	0
2	" "	225	0	0
3	" "	234	0	0
4	" "	234	0	0
5	Streak (1939)	234	0	20
6	" (1941)	234	0	2
7	" "	234	0	30
8	Streak and mosaic (1941)	210	23	11
9	Streak (1941)	117	0	12
10	Mosaic (1940)	350	0	0
11	Virus-free	234	0	0
12	" "	234	0	0

[a] Seed collected the previous season unless otherwise noted.

plants were kept until definite necrotic symptoms appeared and there may have been some opportunity for spread of the virus.

All commercial lots of seed tested carried one virus and some lots carried both viruses. Often both viruses were carried in individual seeds. The streak virus remained active in four-year-old seed, but the mosaic virus was inactivated by one year of storage in seed lots 3 and 4 of commercial seed. In further tests with seed collected at Pullman, Washington, from mosaic-

affected plants (Table 3, plants 8 and 11 in 1941–1942), the mosaic virus was not inactivated in one-year-old seed.

The tests with locally selected seed showed that virus-free seed could be obtained by carefully selecting symptomless plants for seed production (Tables 1 and 3). In 1942–1943, however, selected seed failed to produce healthy plants in greenhouses 7 and 17. Weeds under the benches in these greenhouses appeared to be responsible for the carry-over of the virus from one season to the next. Definite symptoms of spotted wilt were observed on prickly lettuce (*Lactuca scariola*) under the benches in greenhouse 7. Symptoms of a possible virus disease on wood sorrel (*Oxalis pumila*) under benches in greenhouses 7 and 17 also indicated that this weed might be responsible for the carry-over of the virus which could be transmitted readily by thrips to the new crop of cineraria.

MECHANICAL TRANSMISSION

Preliminary tests in 1938–1939 showed that both the streak and mosaic viruses could be successfully transmitted by mechanical inoculation methods.

TABLE 4.—*The effect of extraction of plant juice upon the activity of the streak and mosaic viruses*

Virus used	Method of inoculation	Results obtained in different series of inoculations[a]	Period of incubation
			Days
Streak	Rubbed leaf tissue	7/20, 8/10, 12/20, 8/10, 3/5	14–35
Streak	Juice extract	0/40, 0/20, 0/20[b]	
Mosaic	Rubbed leaf tissue	6/10, 14/20	25–42
Mosaic	Juice extract	13/20	35–42
Controls	No treatment	0/40, 0/10, 0/40, 0/40	

[a] Each fraction represents a series of inoculations, the numerator shows the number of plants infected and the denominator the number of plants inoculated.

[b] Ten cc. of one-half per cent sodium sulphite was added to 100 grams of frozen leaf tissue previous to extraction of juice in press.

Forty to 82 per cent of the mosaic inoculations and 16 to 50 per cent of the streak inoculations were successful. In these tests portions of the diseased leaf were wrapped around a cotton swab and slightly macerated on a pot label before being rubbed over the foliage of the plants to be inoculated. Carborundum dust was sprinkled over the leaves to be inoculated previous to rubbing the diseased tissue upon them.

In later inoculation tests, with 2 other methods of inoculation based on the extraction of the plant juice from the leaves before rubbing onto healthy foliage, the streak virus was not transmitted (Table 4). These methods included macerating the affected leaves in a mortar, filtering through cheesecloth or freezing the affected leaves for 12 hours before extracting the juice in a hydraulic press. In each case the juice was diluted one to 10 with distilled water before being rubbed onto leaves dusted with carborundum. The mosaic virus was transmitted equally well by all of the inoculation methods used (Table 4). The streak virus was inactivated very quickly in

extracted plant juice, and accordingly, the handling of plants in ordinary cultural practices probably would not account for much transmission of the disease. The relative ease of transmitting the mosaic virus in extracted juice indicated that the handling of plants in routine cultural practices might account for considerable transmission of the disease.

INSECT TRANSMISSION

Two insects, *Aphis marutae* Pestlund[6] and *Thrips tabaci* Lindeman, were commonly observed on cineraria plants. These insects were transferred to healthy plants under controlled conditions to determine if they would transmit the viruses (Table 5). The mosaic virus was transmitted by *Aphis marutae* and the streak virus was transmitted by *Thrips tabaci*.

TABLE 5.—*Insect transmission of the mosaic and streak viruses*

Source of virus	Insect tested	Results obtained in different tests[a]
Cineraria streak	*Aphis marutae*	0/6, 0/27, 0/10
Cineraria streak	*Thrips tabaci*	5/5, 12/15
Control	No treatment	0/6, 0/30, 0/10, 0/5, 0/15
Cineraria mosaic	*Aphis marutae*	3/5, 3/5, 6/10, 3/10, 19/37
Cineraria mosaic	*Thrips tabaci*	0/20, 0/5
Controls	No treatment	0/9, 0/5, 0/4, 0/10, 0/25

[a] Each fraction represents a series of inoculations, the numerator shows the number of plants infected, the denominator shows the number of plants inoculated.

THERMAL INACTIVATION AND LONGEVITY

The streak virus was inactivated by extraction from plant tissue in a number of inoculation tests. The thermal inactivation point of the virus could not be determined. It has been reported that the use of sodium sulphite in preparing an extract of chrysanthemum facilitated the detection of the spotted wilt virus in that host.[7] The use of 0.5 per cent solution of anhydrous sodium sulphite in preparing extract of cineraria leaves failed to increase the longevity of the streak virus.

The mosaic virus (Table 6) was inactivated by 10-minute exposure at a point near 70 degrees C., and it remained active in extracted juice for 14 days but not for 100 days.

HOST RANGE

A number of different crops were inoculated to determine the possible relationship of the cineraria viruses to other known viruses (Table 7). The streak virus proved to have many characters associated with the spotted wilt virus, and tomato and pea were inoculated to ascertain if these two viruses were the same. The mosaic virus was compared with pea mosaic as well as being tested on tobacco, tomato, cucumber, and pea. The results of cross

[6] Determination kindly made by Doctor George F. Knowlton, Utah State Agricultural College, Logan, Utah.

[7] Ainsworth, G. Detection of spotted wilt virus in chrysanthemum. Nature, **137**: 868. 1936.

TABLE 6.—*The thermal inactivation point and longevity in vitro of the cineraria mosaic virus*

Treatment of inoculum[a]	Results of inoculations[b]
Leaf tissue	7/10, 6/10, 12/18
Fresh extract	10/20, 6/20, 12/18
Extract—50° C.	3/20, 5/20, 11/18
" —60° C.	6/20, 0/20, 8/18
" —70° C.	5/20, 0/20, 0/18
" —80° C.	0/20
Extract after inoculation	9/20, 5/20
" " 24 hours	13/18
" " 5 days	9/20
" " 10 days	2/20
" " 14 days	8/20
" " 100 days	0/20
Control	0/20, 0/20, 0/36

[a] Extracted juice was exposed to temperatures for 10 minutes.
[b] Each fraction represents a series of inoculations, the numerator shows the number of plants infected, the denominator shows the number of plants inoculated.

inoculations indicate that the mosaic virus is specific to cineraria, because it did not infect any of the other hosts tested. Comparison of the cineraria mosaic virus and the lettuce mosaic virus failed to show any relationship between these two viruses. The streak virus was successfully transmitted to tomato and pea, and the symptoms produced on these hosts further indicated a very close relationship of this virus to the tomato spotted-wilt virus. Tomato plants with symptoms commonly associated with infection by the spotted-wilt virus were used to inoculate cineraria. The symptoms produced on cineraria were similar to those associated with infection by the streak virus. These cross-inoculation tests and other characters, such as rapid

TABLE 7.—*The host range of the cineraria mosaic and streak viruses*

Source of virus and host inoculated	Results obtained in different series of inoculations[a]
Mosaic virus from cineraria:	
Cineraria	33/40, 6/8
Tobacco	0/25
Tomato	0/34, 0/29, 0/10, 0/30
Cucumber	0/10
Lettuce	0/20
Pea	0/12
Streak virus from cineraria:	
Cineraria	1/6, 5/10, 20/40
Tomato	3/30, 4/25
Pea	3/20
Spotted wilt virus from tomato:	
Cineraria	6/20, 6/24
Lettuce mosaic virus from lettuce:	
Cineraria	0/8, 0/14
Lettuce	11/20

[a] All inoculations made by rubbing macerated leaf tissue on plants previously dusted with carborundum. The numerator denotes the number of plants infected and the denominator the number of plants inoculated. In each series a like number of plants were left untreated as controls. No disease was noted in the control plants.

inactivation *in vitro* and transmission by *Thrips tabaci*, show that the two viruses are the same or that the streak virus is a variant of the spotted-wilt virus. The host range of the tomato spotted-wilt virus is very large, the disease having been reported on 84 species in 19 families of the plant kingdom.[8] Since the cineraria streak virus and the tomato spotted-wilt virus are similar in nature, it may be assumed that the former has a host range quite similar to the latter.

CONTROL

Streak and mosaic can be controlled by carefully selecting disease-free plants for seed production. Selected seed was furnished to 3 growers in 1940–1941, 5 growers in 1941–1942, and 6 growers in 1942–1943. In each case in the first 2 years of tests the selected seed produced mosaic- and streak-free stock. In 1942–1943, however, streak developed in two of the greenhouses on plants produced from selected seed. These results indicated the importance of weeds in the carry-over of the virus from one cineraria crop to another and accordingly the destruction of weeds in the greenhouse may be an important control measure. In 1941–1942, two of the growers produced plants from both selected and non-selected seed. In each case streak was evident in the plants from the non-selected seed and was absent in plants from the selected seed. Observations indicate that thrips spread the streak virus very rapidly from plant to plant, so that an excess of seed plants should be selected when the plants are about half grown and further selection from this group should be made as the plants mature. The control of thrips on cinerarias should be of value in reducing the spread of streak. Selected plants should be moved as far as possible from other cinerarias.

In two greenhouses there has been severe spotted-wilt disease on tomato. This could be traced to streak-affected cinerarias in the vicinity of the tomatoes, hence it is not advisable to grow streak-affected cinerarias near tomatoes.

Measures of value in controlling mosaic, beyond the use of disease-free seed, include aphid control and the destruction of affected plants whenever noted. Since the mosaic virus is readily transmitted in plant juice and by mechanical injury of plants, care should be used not to handle mosaic-affected plants in routine cultural practices.

SUMMARY

Two virus diseases, streak and mosaic, have been destructive on cineraria in greenhouses of Washington.

Necrosis of leaves and stems and wilting of plants near blossoming time caused by the streak virus often resulted in a loss of 20 to 50 per cent of the plants.

Mottling of the leaves with light and dark green areas, puckering and malformation of the leaves, and dwarfing of plants caused by the mosaic virus in a few cases resulted in the loss of a low percentage of the plants.

[8] Holmes, Francis O. Handbook of phytopathogenic viruses. 221 pp. Burgess Publishing Company. Minneapolis, Minnesota. 1939.

The streak virus is transmitted in seed, by mechanical rubbing of diseased tissue on healthy tissue, and by *Thrips tabaci,* but is inactivated very quickly in extracted plant juice. The streak virus was transmitted to tomato and pea and the symptoms produced on these suscepts as well as the other characters of the virus show that it is a strain of the tomato spotted-wilt virus.

The mosaic virus is transmitted in seed, by mechanical inoculation methods, and by *Aphis marutae.* This virus is inactivated near 70 degrees C. and remains active in extracted plant juice for 14 days. The mosaic virus was not transmitted to tobacco, tomato, cucumber, lettuce, and pea; and accordingly, it appears to be specific to cineraria.

Streak and mosaic can be controlled by carefully selecting disease-free plants for seed production. The control of aphids and thrips on cineraria plants would aid in reducing spread of the mosaic and streak viruses. The destruction of weeds, that may be hosts of the streak virus, in the vicinity of cineraria plants and care in not handling mosaic plants previous to handling healthy plants are cultural practices of value in reducing losses.

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COMPARISON OF SOIL FUMIGANTS FOR THE CONTROL OF THE ROOT-KNOT NEMATODE

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During the past five years chloropicrin has been the outstanding soil fumigant for root-knot-nematode control. However, because it is disagreeable to work with and relatively high priced, there is need for a chemical which does not possess these disadvantages. With this in mind a number of chemicals which showed promise on the basis of small-scale greenhouse tests were compared with chloropicrin for the control of nematodes on tomatoes grown in two commercial greenhouses, one growing a spring crop and the other a fall crop.

LITERATURE REVIEW

The literature on the effectiveness of chloropicrin for nematode control was recently reviewed by Taylor (8), who reported excellent results with it at 200 pounds per acre. Several workers have obtained satisfactory nematode control with ethylene dichloride, both alone and when mixed with chloropicrin (2, 4, 6). Methyl bromide, an industrial fumigant, has been used as a soil fumigant against insect pests, and in 1940 Taylor and McBeth (9) reported good nematode control with it, using 80 cc. of the chemical per cubic meter of soil. Good results have since been reported by Taylor and McBeth (10), Gingrich and Haenseler (3), and Godfrey and Young (4). These workers all attempted to use undiluted methyl bromide, which necessitates either keeping the chemical below its boiling temperature of 40° F. or using special apparatus for measuring and dispensing it. The use of D–D mixture,[1] a crude by-product from the manufacture of allyl alcohol, containing approximately equal parts of 1,2-dichloropropane and 1,3-dichloropropylene, was first reported by Carter (1), who used it successfully against *Heterodera marioni* in Hawaii. Pinckard (7) also endorsed D–D mixture as a nemacide.

TREATMENTS FOR SPRING TOMATO CROP

The first experiments were in a commercial greenhouse of 10,000 square feet on sandy loam heavily infested with root-knot nematode in Auburn, N. Y.[2] One section of 2,000 square feet of ground bed was divided into 20 plots. The soil was thoroughly prepared with a power-driven rotary tiller. The liquid fumigants were applied with hand-operated injectors, 4 to 5 inches deep, at 10-inch intervals, in rows 10 inches apart. Four replicate randomized plots were treated with each of the following four chemicals and four were left untreated. Chloropicrin,[3] at 2 cc. per injection, was applied

[1] Supplied by the Shell Development Co., Emeryville, California.

[2] Acknowledgment is made to Dickman Brothers for their wholehearted cooperation.

[3] The chloropicrin was supplied by Innis, Speiden and Co., 117 Liberty Street, New York, as "Larvacide."

August 26, at which time many root galls from the previous tomato crop had not yet thoroughly rotted. One week later ethylene dichloride was applied at 15 cc. per injection, and a 10 per cent methyl bromide mixture,[4] at 5 cc. per injection. This methyl bromide mixture containing 67 per cent ethylene dichloride and 23 per cent carbon tetrachloride, which diluents help to keep the methyl bromide in solution, can be used in ordinary applicators. The furyl-nitro-ethylene dust[5] was broadcast at 2 pounds per 100 square feet and was then promptly forked under. After all treatments the soil was leveled by raking and was watered lightly.

After treatment in the fall of 1942 a succession of two or three crops of beet greens was grown. Examination of the roots of the first beet crop grown while the soil temperature was still fairly high gave some indication of the degree of nematode control. Plants from all plots showed some nematode injury. The methyl bromide mixture gave the best control, the chloropicrin and ethylene dichloride being almost as effective. Control in the dust-treated plots was little better than in the untreated ones in which nematode damage was severe. Subsequent beet crops were grown at such low temperatures that no injury occurred.

In March, 1943, another section of 2,000 square feet was divided into 20 plots and treated with the fumigants listed in table 2: namely, chloropicrin, chloropicrin ethylene dichloride (1–9) mixture, chloropicrin methyl bromide (3–1) mixture, and the 10 per cent methyl bromide mixture. This section of the greenhouse had been treated the previous fall with chloropicrin but, since the beet crop indicated that complete nematode control was not obtained, it was decided to re-treat before the spring tomato crop was planted. The soil was mechanically tilled before and was watered immediately after the treatments were made. In order to establish the optimum dosage of the 10 per cent methyl bromide mixture, treatments of 3, 5, and 7 cc. per injection were compared in a third 2,000-square-foot section of the house. Each treatment was replicated four times. Also, in a fourth section, dosage rates of 4, 5, 6, and 7 cc. per injection were employed, each treatment being replicated three times. These two sections had been treated in the fall of 1942, one-half of each with chloropicrin and one-half with furyl-nitro-ethylene dust, with apparently unsatisfactory results. In April the entire greenhouse was planted with tomatoes, variety Break O'Day. Records were kept on the weight and number of ripe fruits obtained from each.

At the end of the growing season the plants were all pulled and their roots examined and scored to indicate the degree of nematode injury. The method of scoring, previously described (6), was to select as standards five individual plants showing various degrees of nematode injury (Fig. 1, A), ranging from no infection, designated zero, to very severe infection, as-

[4] The ethylene dichloride and the 10 per cent methyl bromide mixture (Dowfume Br. 10) were supplied by Dow Chemical Co., Midland, Michigan.

[5] The furyl-nitro-ethylene dust, supplied by Grasselli Chemicals Division of Du Pont, Wilmington, Del., as compound No. IN2391A_2, contained 50 per cent of the active ingredient.

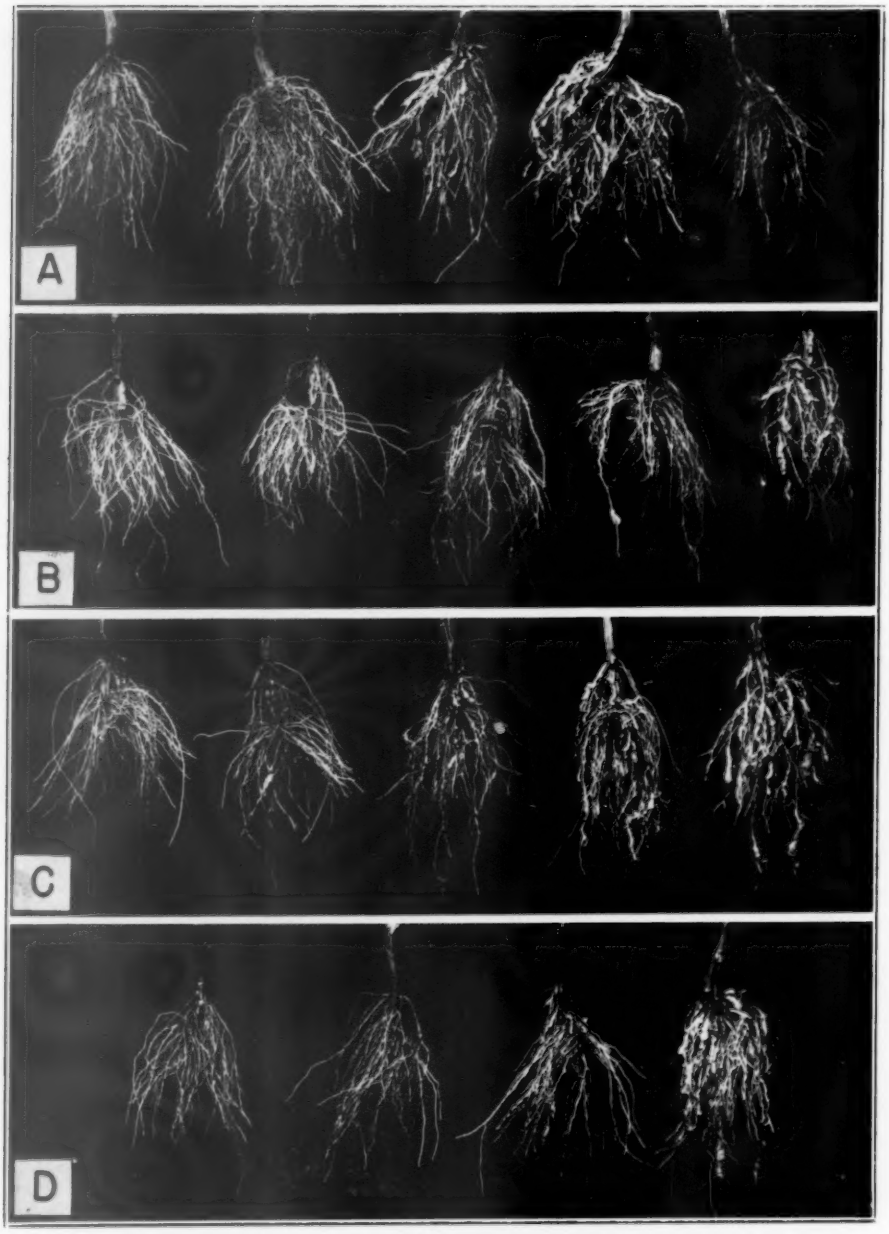

FIG. 1. A. Roots selected from spring crop as standards by which to score those from the experimental plots. They represent (left to right) classes 0, 1, 2, 3, and 4. B. Representative roots from plots receiving (left to right) 10 per cent methyl bromide mixture, 5 cc.; ethylene dichloride, 15 cc.; chloropicrin, 2 cc.; furyl–nitro–ethylene dust (2 lb. per 100 sq. ft.); and no treatment. C. Roots from plots receiving (left to right) chloropicrin–methyl bromide (3–1) mixture, 2 cc.; chloropicrin, 2 cc.; 10 per cent methyl bromide mixture, 5 cc.; chloropicrin–ethylene dichloride (1–9) mixture, 10 cc.; and no treatment. D. Roots from plots receiving 10 per cent methyl bromide treatment at (left to right) 7 cc., 5 cc., 3 cc., and no treatment.

signed a value of 4. The roots from each plot were then scored by comparing them individually with these standards. The scores for each plot were then totaled and divided by the number of roots on that plot. This average value was considered an estimate of the extent of nematode damage. Higher scores indicate greater injury. The plot scores and the yields from each treatment are summarized in tables 1, 2, 3, and 4.

All Fall treatments except the furyl-nitro-ethylene dust gave significant reductions in nematode injury on the spring tomato crop (Table 1 and Fig. 1, B). These reductions were reflected in significantly greater yields. A coefficient of correlation of -0.78 was found between yield and degree of

TABLE 1.—*Effect of treating heavily infested soil with various fumigants on the incidence of root-knot nematode, and the yield and value of a spring tomato crop*

Treatment	Dosage[a]	Ave. root scores	Ave. yield per plant	Ave. weight per fruit	Ave. fruits per plant	Ave. cash income per plant
			Lb.	*Lb.*	*No.*	
Check	0	2.75	4.92	0.35	14.1	$1.15
Furyl-nitro-ethylene dust	2 lb.	2.61	5.03	0.33	15.2	$1.16
Chloropicrin	2 cc.	1.70	6.00	0.36	16.6	$1.36
Ethylene dichloride	15 cc.	1.11	6.23	0.36	17.3	$1.44
Methyl bromide mixture (10%)	5 cc.	0.60	6.00	0.35	17.0	$1.35
Least diff. req. for odds	19:1	0.50	0.64	0.04	1.4	0.21
	99:1	0.70	0.90	0.05	2.0	0.29

[a] With the dust treatment 2 lb. dosage represents amount used per 100 square feet. With the liquids, figures give amount used per injection. Injections 10 inches apart.

nematode injury. Failure of the dust treatment was disappointing, because this material had appeared very effective in small-scale preliminary trials. Possibly, inability to obtain a satisfactory mixing of the dust with the soil was the main cause of its failure under commercial conditions. Use of a mechanical rotary tiller to incorporate it with the soil, however, gave no better results. Better nematode control was obtained with the methyl bromide mixture than with either chloropicrin or ethylene dichloride, but all three produced about the same yields. The relatively ineffective control with chloropicrin may have been due to the fact that treatment was made before nematode galls from the previous crop had thoroughly rotted.

The average cash income per plant was computed by multiplying the yield for each day by the price obtained on that day. Since the nematodes had more effect on the plants in the untreated plots as the season progressed, the greater part of the increase in yield on the treated plots over the check plots occurred near the end of the season, at which time prices were low (Fig. 2). It is obvious, therefore, that multiplying the total yield from each treatment by an average price for the season, as is often done, would have given too high a value to the increased income due to treatment. For

example, if it were assumed that a treated and an untreated plot yielded the same until the last two pickings, at which time the treated plot yielded 5 pounds more than the check, the apparent increase in income would have been $1.25 if the yields for the season were multiplied by the average seasonal price (*i.e.*, 25¢ a pound). However, the actual increase in income, obtained by multiplying by the value of the tomatoes when picked (*i.e.*, 5¢ a pound), would have been only 25¢.

When the price is constant throughout the picking season, as is frequently the case with the fall crop, the use of an average price may be justifiable in computing increased income due to treatments. This was the case in the fall experiment recorded in table 8.

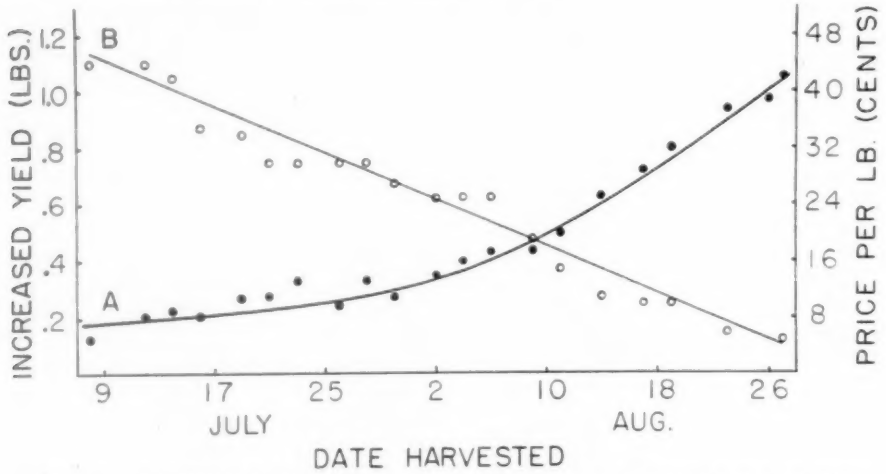

FIG. 2. Cumulative increase in tomato yields (A) (on a per plant basis) of chloropicrin-treated plots over the untreated plots, and price of tomatoes (B) throughout the season. Note that yield (A) is expressed as cumulative or total increase and that greatest increase came at the end of the season when prices were lowest. Ethylene dichloride and the methyl bromide mixture each gave similar curves.

All treatments listed in table 2 significantly reduced nematode injury. However, the nematode damage to the checks was not sufficient to reduce yields to the point where differences could be demonstrated statistically. When one realizes that the yield of the plants in the untreated plots was not markedly reduced, even when the roots were injured to the extent shown in figure 1, C, it is easy to understand why differences in yield between treatments are difficult to demonstrate.

The data in tables 3 and 4 indicate that 3- to 4-cc. injections of the 10 per cent methyl bromide mixture gave sufficient nematode control to permit normal yields of tomatoes. Increasing the dosage to 7 cc. resulted in better nematode control (Fig. 1, D), but yields were not improved. It appears that a low dosage that will reduce the nematode population sufficiently to permit young plants to get a good start will result in satisfactory yields by the current crop. However, if a second high-temperature crop suscep-

TABLE 2.—*Effect of treating lightly infested soil with various fumigants on the incidence of root-knot nematode and the yield and value of a spring tomato crop*

Treatment	Dosage per injection	Ave. root scores	Ave. yield per plant	Ave. weight per fruit	Ave. fruits per plant	Ave. cash income per plant
	Cc.		*Lb.*	*Lb.*	*No.*	
Check	0	2.04	6.87	0.37	18.7	$1.80
Chloropicrin–ethylene dichloride mixture (1–9)	10	0.67	7.23	0.35	20.6	$1.82
Methyl bromide mixture (10%)	5	0.51	7.10	0.36	19.6	$1.79
Chloropicrin	2	0.40	7.38	0.36	20.7	$1.89
Chloropicrin–methyl bromide mixture (3–1)	2	0.14	7.47	0.37	20.1	$1.88
Least diff. req. for odds	19: 1	0.46	0.90	.....	2.05	0.18
	99: 1	0.64	1.26	.....	2.87	0.25

tible to nematodes is to follow the first, then a more effective dosage might be desirable. The value of a low-dosage treatment before each crop as compared with a heavy treatment for two crops remains to be established. The optimum dosage may well depend on the crops grown, as well as amount of inoculum in the soil, soil conditions, etc.

When treatments resulted in an increase in yield this increase was due to the production of more fruits per plant rather than to an increase in the size of the individual fruits (Tables 1, 2, and 3). The production of fewer fruits per plant on the untreated plots was not due to early death of plants, as these were still green at the close of the picking season.

TREATMENTS FOR FALL TOMATO CROP

Another series of soil treatments for the control of the root-knot nematode was made in a large commercial greenhouse at Schenectady, N. Y.[6] Treatments were made on June 25 in raised beds from which tomato plants,

TABLE 3.—*Effect of dosage of the 10 per cent methyl bromide mixture on the incidence of root-knot nematode, and the yield and value of a spring tomato crop*

Dosage per injection	Ave. root scores	Ave. yield per plant	Ave. weight per fruit	Ave. fruits per plant	Ave. cash income per plant
Cc.		*Lb.*	*Lb.*	*No.*	
0	2.87	5.02	0.37	13.5	$1.18
3	1.80	5.66	0.37	15.6	$1.37
5	0.86	5.69	0.39	14.4	$1.38
7	0.51	5.91	0.39	15.3	$1.37
Least diff. req. for odds					
19: 1	1.40	0.90	.....	2.2	0.19
99: 1	1.71	1.10	.....	3.3	0.28

[6] Acknowledgment is made to the Brownsey and Marks Greenhouse Company for their cooperation.

TABLE 4.—*Effect of dosage of the 10 per cent methyl bromide mixture on the incidence of root-knot nematode, yield, and value of a spring tomato crop*

Dosage	Ave. root score	Ave. yield per plant	Ave. income per plant
Cc.		*Lb.*	
0	3.65	3.64	$1.00
4	1.69	5.21	$1.21
5	1.75	4.64	$1.34
6	1.31	5.69	$1.22
7	1.48	4.18	$1.43
Least diff. req. for odds			
19:1	0.60	1.09	0.30
99:1	0.87	1.59	0.44

heavily infested with nematodes, had been removed about June 1. The soil was loosened by forking just before treatment. Each of the treatments listed in table 5 was replicated five times in randomized plots. The dosage applied per injection was computed by dividing the total amount of fumigant used on the 5 replicates by the total number of injections made. About the first of August, 14 tomato plants of the variety Vetomold were set in each plot. Records were kept by the grower of the yields from each plot at each picking. At the end of the growing season the authors examined and scored the roots of each plant. The 6 standard roots used for scoring in this experiment are illustrated in figure 3. The results are in table 5.

All treatments gave very satisfactory nematode control. No significant differences in control were obtained between any of them, although the 10 per cent methyl bromide mixture appeared most effective, at the three rates employed, the chloropicrin alone and chloropicrin mixtures came next, and the D–D mixture was the poorest. All treated plots produced more fruit than the untreated, but none of the differences in yield between the treated plots was significant. The low dosage rates of the 10 per cent methyl

TABLE 5.—*Effect of treating nematode-infested soil with various fumigants on the incidence of root-knot nematode and the yield of a fall tomato crop*

Fumigant	Dosage per injection	Ave. root score	Ave. yield per plant
	Cc.		*Lb.*
Check	0.0	2.81	4.54
Methyl bromide mixture (10%)	4.19	0.70	5.65
" " " "	6.16	0.67	5.38
" " " "	8.64	0.76	5.47
Chloropicrin	1.61	0.95	5.50
"	2.58	0.98	5.67
Chloropicrin–ethylene dichloride mixture (1–9)	7.35	0.99	5.35
Chloropicrin–methyl bromide mixture (3–1)	1.80	0.96	5.80
D–D mixture	4.18	1.25	5.45
Least diff. req. for odds 19:1	.....	0.72	0.66
99:1	.....	0.97	0.89

FIG. 3. Roots selected from fall crop as standards by which to score those from the experimental plots. They represent (left to right) classes 5, 4, 3, 2, 1, and 0.

bromide mixture (4.19 cc.) and of the straight chloropicrin (1.61 cc.) resulted in as good yields as treatments at the higher rates of 8.64 and 2.58 cc., respectively. The addition of methyl bromide to chloropicrin appeared more effective than either chloropicrin alone or the chloropicrin–ethylene dichloride mixture. The lower effectiveness of D–D mixture at the 4-cc. rate (6 cc. per square foot) employed was not anticipated in view of results obtained elsewhere with much lower dosages. Possibly this can be attributed to differences in soil type or organic content.

The cost per 1,000 square feet of the various chemicals and mixtures used in these experiments is in table 6. The cost of application is excluded, since this is approximately the same for all of these materials. Differences in total costs of treatments are chiefly due to the prices and quantities of the chemicals used. Application costs may vary from 25¢ to 75¢ per 1,000 square feet, depending on the labor costs, size of area treated, equipment

TABLE 6.—*Cost of the chemicals used for soil fumigation*

Fumigant	Cost per pound	Approx. No. of cc. per pound	Amount per injection on 10-in. centers		Pounds required to treat 1000 sq. ft.		Approx. cost of chemicals to treat 1000 sq. ft.	
			Min.	Max.	Min.	Max.	Min.[b]	Max.[c]
	Cents		*Cc.*	*Cc.*				
D–D mixture	11–15[a]	384	1.0	4.0	3.8	15.0	$0.42	$2.25
Methyl bromide mixture (10%)	16–20	329	3.0	9.0	13.2	39.5	$2.10	$7.90
Ethylene dichloride	8–10	361	10.0	15.0	39.9	59.9	$3.20	$6.00
Chloropicrin–ethylene dichloride mixture (1–9)	16–18	350	7.5	10.0	30.9	41.2	$4.95	$7.40
Chloropicrin–methyl bromide mixture (3–1)	78–83	271	1.5	2.0	8.0	10.6	$6.25	$8.80
Chloropicrin	80–85	274	1.5	2.5	7.9	13.1	$6.30	$11.15
Methyl bromide	70–75	262						

[a] Limited availability. Market price not yet fixed.
[b] Calculated from figures for lowest cost and minimum dosage.
[c] Calculated from figures for highest cost and maximum dosage.

available, etc. Thus, total cost of treatment ranges from about $2.50 to almost $10.00 per 1,000 square feet, which in general is well below the range recorded by Newhall (5) for steaming in commercial greenhouses ($6.50 to $20.00 per 1,000 square feet).

The data in table 7, showing economic value of treatments for greenhouse tomatoes on an individual plant basis, computed from results on the spring crop, and in table 8, computed from the fall experiment, indicate the relatively low cost of treatment in relation to the income from the crop.

TABLE 7.—*Economic value of soil treatments in a tomato greenhouse on a per plant basis. Spring of 1943*

Fumigant	Cash income[a]	Cost to treat	Increased income
		Cents	*Cents*
Check	$1.14	...	...
Methyl bromide mixture (10%)	$1.35	1.8	18.2
Chloropicrin	$1.36	3.7	17.6
Ethylene dichloride	$1.44	2.5	26.8

[a] Values taken from table 1.

TABLE 8.—*Economic value of soil treatments in a tomato greenhouse on a per plant basis. Fall of 1943*

Fumigant	Dosage	Cash income[a]	Cost to treat	Increased income
	Cc.		*Cents*	*Cents*
Check	...	$1.13	...	...
Methyl bromide mixture (10%)	4.2	$1.41	1.0	26.8
" " " "	6.2	$1.34	1.5	19.5
" " " "	8.6	$1.37	2.1	21.2
Chloropicrin	1.6	$1.37	2.3	21.7
"	2.6	$1.42	3.8	24.5
Chloropicrin–ethylene dichloride mixture (1–9)	7.4	$1.34	1.6	18.7
Chloropicrin–methyl bromide mixture (3–1)	1.8	$1.45	2.6	28.9
D–D mixture	4.2	$1.36	0.7	22.1

[a] Since the price did not vary materially throughout the picking season on this crop the income was computed by multiplying the yield (Table 5) by the average price for the season (25 cents a pound).

Effectiveness of treatment should be the primary consideration with high value crops, particularly in periods of high prices. While it is realized that these income figures may be somewhat high, nevertheless, even in normal times, the value of most greenhouse crops is sufficiently high to require only slight increase in yield to pay for the cost of treatment. For low value, or outdoor crops, the cost of treatment may become the determining factor in selecting a treatment.

DISCUSSION

The ideal soil fumigant for treatment of greenhouse soils should possess certain characteristics. It should be effective against all types of soil-borne

pathogens and weed seeds. The fumes should not be toxic, irritating or uncomfortable to work in. The vapor escaping from the soil should not be so phytocidal as to prohibit treating areas adjacent to growing plants. The fumigant should be able to successfully penetrate heavy soils and unrotted roots therein. The vapor should escape rapidly enough from the soil or be sufficiently nontoxic to growing plants to permit planting a few days after treatment. Materials should be readily available, low-priced, and noncorrosive to metals.

No one fumigant can be said to be the best in all instances, because the relative importance of the characteristics just enumerated varies under different circumstances. Chloropicrin appears more effective against a wider range of soil pests and weed seeds than any of the fumigants tested. Methyl bromide has some fungicidal and herbicidal value, though probably less than chloropicrin at the concentrations recommended for nematode control. Ethylene dichloride appears to have relatively little value as a fungicide or herbicide. The merits of D–D mixture in these respects remain to be established. It appears, therefore, that if the control of certain fungi or weeds is important, chloropicrin treatment would probably be the most satisfactory.

If nematode control is the primary purpose of soil fumigation, other fumigants have certain advantages over chloropicrin. If a grower wants to replant as soon as possible, methyl bromide mixture is more satisfactory, since it appears to penetrate unrotted roots, and it escapes from the soil quickly so that replanting can take place a few days after treatment. The efficacy of chloropicrin may be reduced if root galls from the previous crop are not given sufficient time to decay before the treatment is made. No information on the penetrating power of ethylene dichloride or D–D mixture into unrotted roots has been obtained. Ethylene dichloride dissipates from the soil fairly rapidly. Toxic quantities of D–D mixture appear to persist in the soil for even longer periods than will chloropicrin. Soil conditions greatly influence the length of retention of any of these chemicals, but their rate of dissipation seems to be in the order indicated.

The lachrymatory effects of chloropicrin make it disagreeable, but it is safe to work with. Methyl bromide mixtures are not disagreeable to handle, but may be somewhat dangerous because of their toxicity to humans. If proper precautions are observed, harmful effects appear unlikely. Neither ethylene dichloride nor the D–D mixture are seriously toxic or especially disagreeable, but breathing vapor of D–D mixture for long periods may have a nauseating effect, and both chloropicrin and D–D may cause chemical burns if left in contact with the skin.

Chloropicrin vapor is very toxic to foliage, which necessitates removing all plants from a house before it may be safely treated. Plants appear more tolerant to the other fumigants, which means that under certain circumstances one area in a greenhouse may be treated without removing plants from nearby areas.

Cost of treatment is not an extremely important factor in selecting a material if high-value crops are to be grown, but the low costs of D–D and the methyl bromide mixture permit their use for low-value crops.

Neither the methyl bromide mixture nor ethylene dichloride have a marked corrosive effect on metals. Consequently, these chemicals can be applied with certain applicators which would be unusable with more corrosive materials, as chloropicrin and D–D mixture.

The advantage of using mixtures of fumigants is often questionable. Adding methyl bromide to chloropicrin results in a mixture which has good penetrating power and is fungicidal, but the mixture is more difficult to handle than chloropicrin alone and the lachrymating and corrosive features of chloropicrin are still present. Mixing ethylene dichloride with chloropicrin results in a more economical fumigant than chloropicrin, but it is still disagreeable to use and the effectiveness seems to be proportional to the amount of chloropicrin. On the other hand, mixing methyl bromide with other chemicals is necessary to keep it from vaporizing too rapidly at ordinary temperatures. Methyl bromide mixtures other than the ones used in these experiments may prove more valuable as soil fumigants.

The chief advantages of soil fumigation with chemicals over heat treatment, such as steam or hot water, lie in their greater economy and lower equipment and labor requirements. Steam treatment, however, is more effective against a wider range of soil pests, can more safely be made in houses containing growing plants, and usually permits the soil to be replanted more promptly.

Hand injectors were employed in these experiments: a "Larvajector,"[7] model RR for chloropicrin, model A for D–D and a "Mack's Chemical Injector"[8] for other fumigants. In more recent tests, still under progress, a two-row, continuous-flow, power-driven type of applicator has been used in comparison with hand-operated injectors. Use of such apparatus reduces labor and time, thus adding to the practicability of fumigation as a method of controlling soil-borne pests.

SUMMARY

The efficacies of several soil fumigants against *Heterodera marioni* were compared in two commercial tomato greenhouses. Control of root knot was measured by comparing yields and by scoring all roots on several series of replicated plots.

The following materials, listed alphabetically, and rates of application gave good control when injected 4–5 inches deep on 10-inch centers:

Chloropicrin (Larvacide), used at 1.6, 2, and 2.5 cc. per injection.

Chloropicrin plus ethylene dichloride (1 to 9) at 7.35 and 10 cc. per injection.

[7] Procured from Innis, Speiden and Co., Niagara Falls, N. Y. Delivers up to 4 or 5 cc. per injection.

[8] Procured from Stauffer Chemical Co., New York. Delivers up to 60 cc. per injection.

Chloropicrin plus methyl bromide (3 to 1) at 1.8 and 2.0 cc. per injection.

D–D mixture, containing approximately equal parts of 1,2-dichloropropane and 1,3-dichloropropylene, used at 4 cc. per injection.

Ethylene dichloride at 15 cc.

Methyl bromide mixture (Dowfume Br. 10), containing 10 per cent methyl bromide in a 3 to 1 mixture of ethylene dichloride and carbon tetrachloride, used at 3, 4, 5, 6, 7, and 8 cc. per injection.

The advantages and disadvantages of each are discussed. Furyl-nitroethylene dust at 20 pounds per thousand square feet failed to control root knot.

Increasing the dosage of chloropicrin above 1.6 cc. and of the 10 per cent methyl bromide mixture above 3 cc. per injection did not result in larger yields, even though more complete control of nematodes was obtained.

Increased yields from the treated plots were due to the production of more tomato fruits, rather than to an increase in their average size.

Certain of the materials have advantages over chloropicrin for nematode control either because they are cheaper, easier to handle, or permit replanting sooner. The 10 per cent methyl bromide mixture appears very promising in all these respects.

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THE USE OF FUNGICIDES AND GROWTH SUBSTANCES IN THE CONTROL OF FUSARIUM SCALE ROT OF LILIES

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Easter lilies (*Lilium longiflorum* Thunb.) are propagated from bulb offsets, stem bulblets, and scales. These last are fleshy overlapping segments of the bulb and afford the most rapid means of increasing desired types. However, when the scales are removed from the bulbs their injured bases are vulnerable to attack by the basal rot organism, *Fusarium oxysporum* f. *lilii* Imle. Some of the new Easter lily clons developed at the Plant Industry Station, Beltsville, Maryland, are very susceptible to this rot. Imle (9) states that the culture of many other lily species, such as *L. auratum* Lindl., *L. Browni* Poit., *L. bulbiferum* L., *L. candidum* L., *L. formosanum* Stapf, *L. speciosum* Thunb., *L. testaceum* Lindl., and *L. Willmottiae* Wils., is limited by this disease.

In an effort to find a suitable fungicide which could be applied to scales for protection against rot, a number of materials and combinations of materials have been tested on scales of *Lilium longiflorum* and *L. testaceum*. Certain of these fungicides controlled Fusarium rot and in addition some of them stimulated root and shoot growth when used alone or as carriers for synthetic growth substances.

MATERIALS

Lilium longiflorum and *L. testaceum* bulbs used in these tests were grown at the Plant Industry Station, Beltsville, Maryland, in 1943. After harvest the bulbs were packed in moist peat and held in common storage until used.

The method of handling the scales in greenhouse tests has been described in detail elsewhere (10, 14). Essentially it consists in removing the scales from the bulbs, treating them with growth substances, placing them on sand on greenhouse benches, and covering with 2 to 3 inches of wet sphagnum moss. Bulblets and roots form in from 3 to 6 weeks, and the scales may then be planted in flats or in the field.

The following materials were employed in the scale treatments: Fermate (ferric dimethyldithiocarbamate, Du Pont Semesan Co.); Zincate (zinc dimethyldithiocarbamate, R. T. Vanderbilt Co.); Spergon (98 per cent tetrachloro-para-benzoquinone plus 1 per cent of a buffering agent, U. S. Rubber Co.); New Improved Ceresan[3] (5 per cent ethyl mercury phosphate, Du Pont Semesan Co.); Arasan (50 per cent tetramethyl thiuramdisulfide, Du Pont Semesan Co.); 37 to 40 per cent formalin solution; naphthaleneacetic acid; and indolebutyric acid.

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[3] Hereafter in this paper New Improved Ceresan will be referred to as Ceresan.

METHODS AND RESULTS

Experiment 1. Scales from bulbs of a clon, No. 224–34, which had been very susceptible to *Fusarium* in the field, were treated as follows: Some were dipped momentarily in a suspension of Fermate or of Zincate, each at the rate of 1 pound to 100 gallons of water; others were dipped for 2 minutes in a suspension of Ceresan at the rate of 1 pound to 40 gallons of water; a fourth lot was coated with Arasan at the rate of 2 ounces to 60 pounds of scales; a fifth lot was coated with Spergon at the rate of 2 ounces per 100 pounds of scales; and a sixth lot was soaked for 30 minutes in a 2 per cent formaldehyde solution (1:50 dilution of commercial formalin). The Spergon and Arasan were applied by shaking the weighed scales in a paper sack containing the required amount of material. Approximately 150 scales were treated with each fungicide, then separated into 3 lots, placed in 3 different plots on a greenhouse bench, and covered with sphagnum.

Thirty-three days later the scales were examined and photographed. Those treated with Spergon or Arasan were protected from scale rot and had by far the greatest development of roots, bulblets, and shoots (Fig. 1). Ceresan afforded excellent protection from rot but inhibited bulblet formation. Scales treated with formaldehyde showed considerable surface injury. Fermate and Zincate were relatively ineffective as protectants.[4]

Experiment 2. Growth stimulation resulting from Arasan and Spergon in Experiment 1 suggested that if these materials were combined with Ceresan the inhibiting effect of the latter might be overcome. It also seemed desirable to test combinations of each fungicide with a number of root-inducing chemicals that have been recommended as aids in propagation (10). The latter furnish no protection against rot and may cause injury to the scales.

A second experiment was set up in which the treatments included combinations of fungicides and growth substances. Two types of scales were used. The first type was from a mixture of Easter lily clons that were apparently free of Fusarium rot when dug and scaled. The second type was from a Fusarium-susceptible clon, No. 38. No scales showing evidence of disease were used. The following treatments were applied to scales of each type: Ceresan, Spergon, Arasan, naphthaleneacetic acid 1:5000 in talc, indolebutyric acid 1:5000 in talc, 2 per cent formaldehyde, a 30-minute water soak, and an untreated control. The following combinations were also used: Ceresan and Spergon, Ceresan and Arasan, Spergon and naphthaleneacetic acid, Spergon and indolebutyric acid, Arasan and naphthaleneacetic acid, Arasan and indolebutyric acid, 2 per cent formaldehyde solution containing 0.001 per cent naphthaleneacetic acid, and 2 per cent formaldehyde solution containing 0.001 per cent indolebutyric acid. Scales were treated with Ceresan as in Experiment 1. When Arasan or Spergon were used either alone or as carriers for the growth substances, weighed amounts of the powders were not used but the scales were coated by shaking them in

[4] In more recent tests Fermate dusted on lily scales has provided excellent protection against rot.

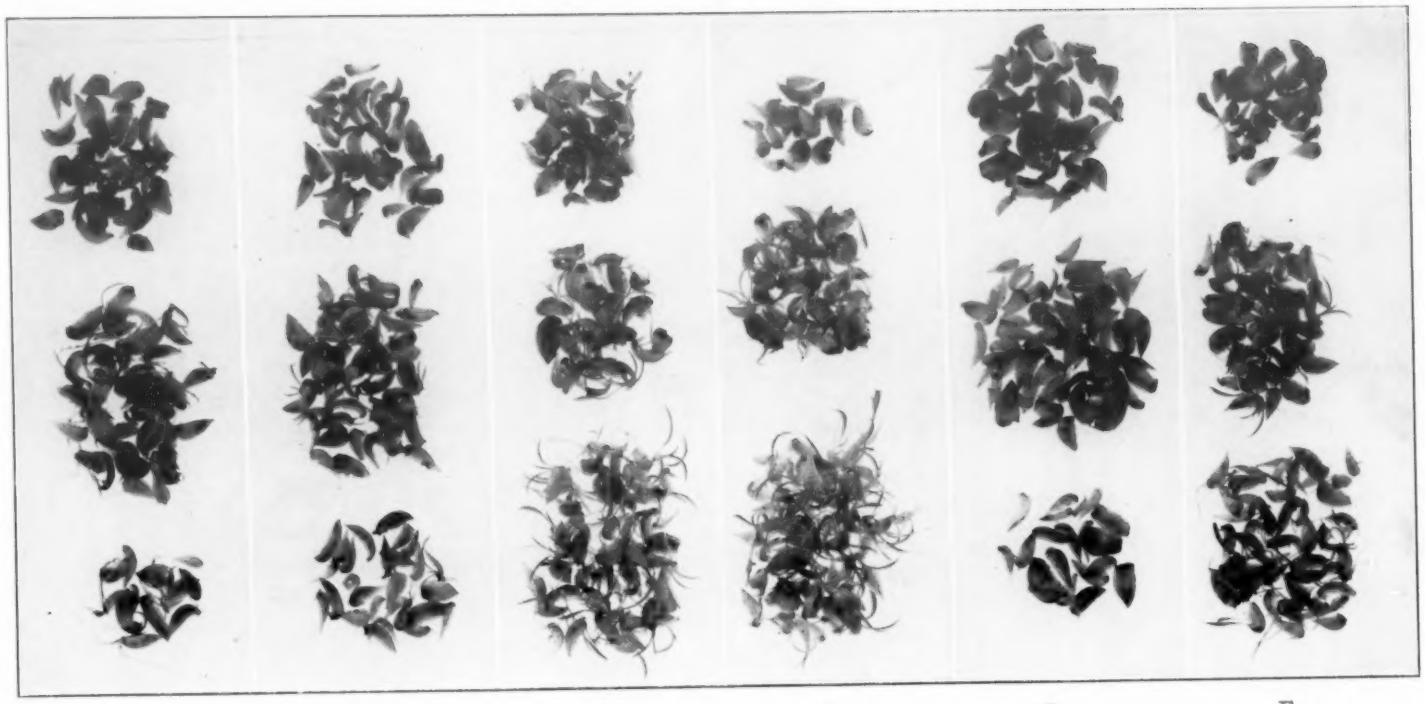

FIG. 1. Fusarium-susceptible Easter lily scales 33 days after treatment with the following materials: A, Fermate; B, Zineate; C, Spergon; D, Arasan; E, New Improved Ceresan; and F, formaldehyde. Upper row (all treatments), infected scales without bulblets; middle row (with the exception of treatment E), infected scales with bulblets; and lower row, healthy scales, with and without bulblets. Middle group of treatment E, healthy scales without bulblets; and lower group of E, healthy scales with bulblets. There were approximately 150 scales in each treatment. Those scales that were entirely rotted are not included.

a paper bag with an excess of these materials. When Ceresan was used with Spergon or with Arasan the scales were first dipped for 2 minutes in a suspension of 1 pound of the mercurial to 40 gallons of water, allowed to dry, then dusted with Arasan or Spergon. In all other treatments in which combinations were used the materials were combined prior to application. Scales were soaked for 30 minutes in the formaldehyde solutions.

The naphthaleneacetic and indolebutyric acid mixtures were prepared at a concentration of 1 part in 5000 parts of talc, Spergon, or Arasan. The acids were dissolved in sufficient 95 per cent ethyl alcohol to wet thoroughly

TABLE 1.—*Numbers of healthy scales, scales with bulblets, and bulblets on two groups of Easter lily scales 40 days after treatment with fungicides and growth substances. One hundred and fifty scales from each group were used for each treatment*

Treatment	Mixture of clons[a]			Clon No. 38		
	Healthy scales	Scales with bulblets	Bulblets	Healthy scales	Scales with bulblets	Bulblets
	Number	*Number*	*Number*	*Number*	*Number*	*Number*
New Improved Ceresan	122	110	154	84	62	85
Spergon	109	144	242	67	111	168
Arasan	129	148	258	103	124	184
Naphthaleneacetic acid 1:5000	26	119	197	32	95	140
Indolebutyric acid 1:5000	23	109	165	19	59	92
New Improved Ceresan + Spergon	129	94	153	96	65	86
New Improved Ceresan + Arasan	140	142	287	125	126	207
Spergon + naphthaleneacetic acid 1:5000	110	145	266	61	116	189
Spergon + indolebutyric acid 1:5000	111	144	267	68	101	138
Arasan + naphthaleneacetic acid 1:5000	127	149	276	116	129	227
Arasan + indolebutyric acid 1:5000	134	145	268	120	130	212
2% formaldehyde + 0.001% naphthaleneacetic acid[b]	17	130	223	3	71	106
2% formaldehyde + 0.001% indolebutyric acid[b]	3	118	210	0	77	115
2% formaldehyde[b]	5	124	217	9	79	111
Untreated, dry	51	134	199	22	60	74
Untreated, water 30 minutes	32	130	215	17	66	82
Least difference necessary for significance between treatment totals with odds of						
19:1	19.6	12.5	36.9	20.6	25.7	35.5
99:1	26.4	16.8	49.7	27.8	34.6	47.4

[a] Bulbs from these clons appeared to be healthy when scaled.
[b] Scales were injured by the formaldehyde treatment.

the talc or fungicide used as a carrier. After the alcohol had evaporated the mixtures were broken up and stored in darkness.

In each treatment there were 150 scales from clon No. 38 and 150 from the mixed clons. They were divided into lots of 50 each and placed at 3 locations in a greenhouse bench. All scales were covered with sphagnum that was kept moist. The temperature in the greenhouse was 65° to 70° F. during the day and 60° to 65° at night.

The scales were treated October 21, 1943, and examined November 30,

1943. The scales from each treatment were sorted into 2 groups depending upon the presence or absence of bulblets. Each group was further subdivided according to presence or absence of Fusarium infection of the scales. The healthy scales, scales healthy but without bulblets, total scales with bulblets, and total bulblets were counted. These data, except for the number of scales healthy but without bulblets, are in table 1. Scales entirely rotted are not included in the tabulation. It is quite possible that healthy bulblets could be produced from many of the scales not classed as healthy, since lesions frequently were present only at the tips or edges of scales farthest from bulblet production areas. *Fusarium* was the only organism obtained from numerous isolations made from the decaying areas of these scales.

All materials and combinations except naphthaleneacetic acid, indolebutyric acid, and formaldehyde afforded very good protection from *Fusarium*. Of the materials tested individually, Arasan provided the best protection; combined with Ceresan it was even more effective. Arasan and all the combinations in which it was used provided much better protection than did Spergon alone or in the same combinations. The differences between Arasan and Spergon were much greater when used on the Fusarium-susceptible clon No. 38 than when used on the less susceptible mixture of clons. Scales treated with indolebutyric acid or naphthaleneacetic acid did not appear to be injured, but those treated with formaldehyde, alone or in combination with either of these 2 growth substances, were injured. This formaldehyde treatment has been recommended for use on lily bulbs for the control of Fusarium basal rot (9), but it appears to be too severe for use on lily scales.

As in the first experiment, Ceresan inhibited bulblet formation. Seventy of the 300 scales treated with Ceresan in Experiment 2 had not developed bulblets at the time records were taken. In a similar lot of scales treated with Ceresan followed by Arasan only 19 lacked bulblets. On the other hand there were 92 scales without bulblets when Spergon followed the Ceresan treatment. In the other treatments, except the Spergon-indolebutyric acid combination, there were very few (0 to 16) scales without bulblets. Some of the scales from the mixed clons given the Ceresan, Arasan, or Ceresan-Arasan treatments as well as some of the scales not treated were removed from the sphagnum and planted in flats of soil in the greenhouse December 6, 1943. On February 8, 1944, the scales were removed from the soil and photographed (Fig. 2).

Whenever Arasan or Spergon was used, with the exception of the Spergon-Ceresan combination, there were significantly more bulblets than on untreated scales. Arasan treatments yielded more bulblets than the Spergon treatments. Naphthaleneacetic acid in talc applied to scales of clon No. 38 was the only other treatment that resulted in the production of significantly more bulblets than the untreated checks. The greatest root and shoot development and bulblet production occurred when Arasan was used as the carrier for naphthaleneacetic acid (Fig. 3). In general, at the concentra-

tions used, naphthaleneacetic acid resulted in the production of more bulblets than did indolebutyric acid.

Experiment 3. Bulbs of *Lilium testaceum* badly infected with *Fusarium* were scaled and treated with Arasan on November 20. Before treatment the

Fig. 2. Easter lily scales from a mixture of clons 110 days after treatment with New Improved Ceresan (upper row), New Improved Ceresan followed by Arasan (second row), Arasan (third row), and untreated (lower row). Arasan in combination has overcome the inhibiting effects of the Ceresan. Note the rot in the untreated scales.

most severely rotted scales were discarded and the rotted areas of the others removed with a knife. When the scales were examined December 18, 176 of the 189 treated scales (93 per cent) showed no sign of rot whereas only 17 of the 62 untreated ones (27 per cent) were free of decaying areas. There were

274 bulblets (1.4 per scale) on the treated scales compared with but 35 (0.6 per scale) on those untreated.

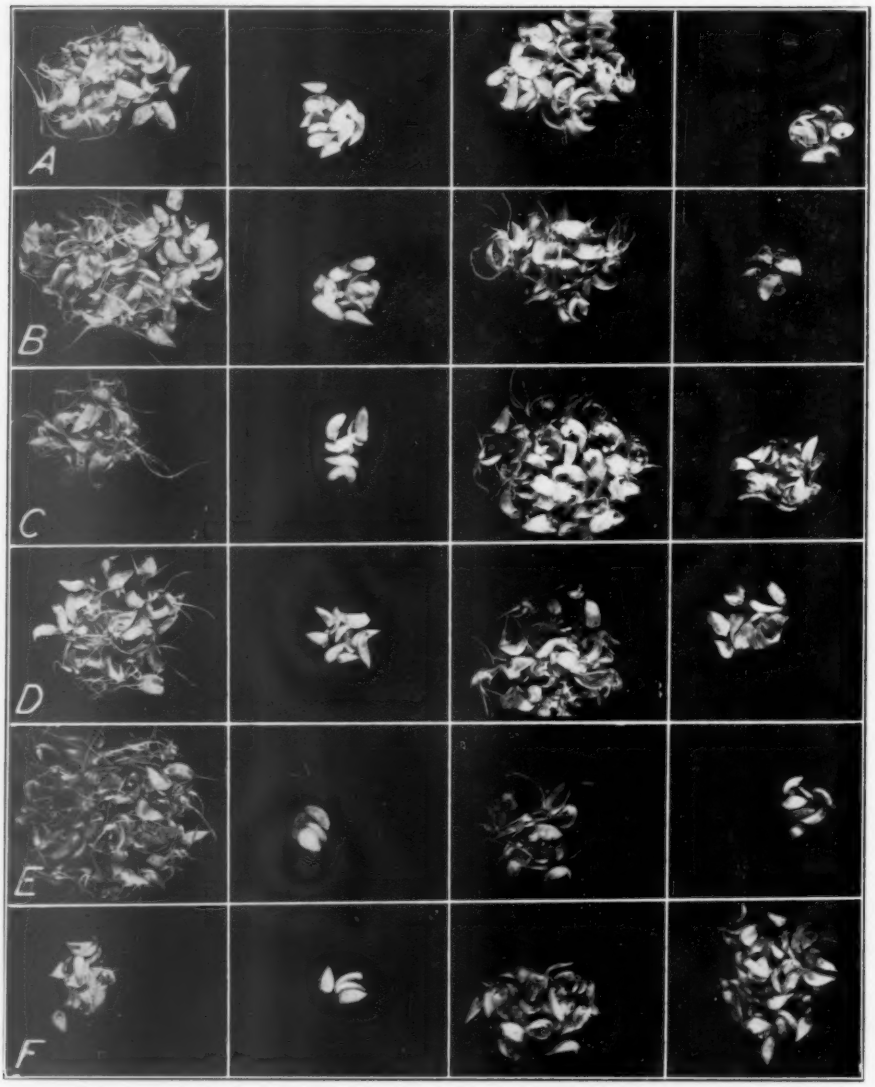

FIG. 3. Bulb scales from a mixture of Easter lily clons 40 days after the following treatments: A, Spergon alone; B, Arasan alone; C, naphthaleneacetic acid alone; D, Spergon as the carrier for naphthaleneacetic acid; E, Arasan as the carrier for naphthaleneacetic acid; and F, untreated checks. One hundred and fifty scales were used in each treatment. From left to right the groups are: healthy scales with bulblets, healthy scales without bulblets, diseased scales with bulblets, and diseased scales without bulblets. Those scales that were entirely rotted are not included.

DISCUSSION

Mercurials and other fungicides have been widely tested as seed protectants. Increased yields resulting from such treatments have been as-

cribed for the most part to better protection of the treated seed during pre- and post-emergence. However, McNew reported evidence for stimulation of pea growth following treatment with Spergon (11).

In the present study Spergon was very effective in protecting Easter lily scales from Fusarium rot during propagation. It was inferior, however, to Arasan in protective action when scales from a very susceptible clon were used. Both fungicides exerted a marked growth-stimulating effect on the treated scales. Part of this stimulation undoubtedly arose from the fact that the Fusarium infection was reduced, but there appears to be more growth acceleration than could be accounted for by this action alone. It has been suggested that the stimulation from the Arasan occurs because of its nitrogen content. This possibility remains to be established. The growth-stimulating properties of Spergon must be explained in some other manner since it contains no nitrogen.

Ceresan afforded good protection against *Fusarium* on lily scales. Its use inhibited bulblet and root formation. This inhibition was largely overcome by treatment with Arasan, thus offering further evidence of the growth stimulating properties of the latter compound. Spergon was ineffective in overcoming Ceresan inhibition. Growth inhibition of lily bulbs following treatment with mercurials has been reported (13).

Growth substances and fungicides recently have been combined for use in seed treatment tests with conflicting results (1, 2, 3, 12, 15). Grace (5, 6) and Grace and Farrar (8), who used ethyl mercury phosphate combined in talc dusts with indoleacetic and naphthaleneacetic acids on woody and herbaceous cuttings, report occasional beneficial results from the combinations. Ceresan (ethyl mercury phosphate) was not combined in the present study with either naphthaleneacetic acid or indolebutyric acid. However, when these 2 growth substances were combined with either Spergon or Arasan, bulblet formation was increased. The effect of the growth substances was expressed in much heavier rooting of the treated scales. The temporary reduction in growth of the bulblets and leaves which is often associated with heavy root production was eliminated by Arasan or Spergon. Addition of naphthaleneacetic acid or indolebutyric acid to formaldehyde did not result in greater bulblet production nor did their addition reduce the injurious effect of the formaldehyde as has been reported for wheat seed by Grace (4, 7). He added naphthaleneacetic acid and indoleacetic acid to formaldehyde or treated the seeds with the growth substances some time after the formaldehyde treatment and was able by either method to overcome the injurious effect of the formaldehyde.

No evidence of incompatibility between growth substances and fungicides has been reported previously or found in the present studies. The effect of other concentrations of growth substances in combination with these and other fungicides needs to be investigated. Such combinations may prove superior to existing preparations for the propagation of many plant materials owing to the provision for disease protection and growth stimulation

over long periods of time. Further investigations of this problem are in progress.

SUMMARY

Bulb scales of two *Lilium longiflorum* Thunb. clons and scales of *L. testaceum* Lindl., all known to be very susceptible to rot caused by *Fusarium oxysporum* f. *lilii* Imle, and scales of a mixture of less susceptible Easter lily clons were treated with some or all of the following fungicides: Ceresan, Arasan, Fermate, Zincate, Spergon, and formaldehyde. The scales were examined after being planted for 4 to 6 weeks on a greenhouse bench under moist sphagnum. Very good protection was obtained with Arasan and Spergon. Arasan was superior to Spergon on the very susceptible scales. In addition, these 2 materials stimulated production of roots, bulblets, and shoots. Fermate and Zincate were relatively ineffective, and formaldehyde injured the scales. Good protection was obtained with Ceresan but bulblet production was inhibited. When Arasan followed the Ceresan treatment this inhibition was overcome. Spergon did not have this effect.

Arasan and Spergon were used as carriers for the growth substances, indolebutyric acid and naphthaleneacetic acid, at 1 part of growth substance in 5000 parts of fungicide. These combinations produced heavier rooting and more bulblets than did the growth substances or fungicides when used alone. Under the conditions of these tests Arasan was superior to Spergon and naphthaleneacetic acid was better than indolebutyric acid.

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14. Stuart, Neil W. Propagating lily scales in sphagnum. Flor. Rev. **93**(2410): 21–22. 1944.
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PHYTOPATHOLOGICAL NOTES

Foliage Infection of Lycopersicon esculentum by Colletotrichum phomoides.—The tomato anthracnose fungus, *Colletotrichum phomoides* (Sacc.) Chester, generally is believed to be pathogenic only on mature, or nearly mature, tomato fruits; although at least two workers have reported leaflet and stem infection. According to Rolfs[1] "Anthracnose appears as small brown spots on the stems and leaves . . . these spots enlarge and the greater portion of the leaves may become invaded." Giddings[2] reported, "Stems may be attacked and in some cases the sunken spots may result in considerable damage." These reports were not supported by experimental evidence and apparently have not been generally accepted. The data reported herein demonstrate that under certain conditions *C. phomoides* may infect tomato foliage.

Seedling tomato plants of the varieties Rutgers, Marglobe, Early Baltimore, John Baer, Chicago, and Garden State were grown until 3 to 5 inches tall. Suspensions of spores, sclerotia-like bodies, and mycelium obtained from agar cultures of *Colletotrichum phomoides* were atomized on the plants. Inoculated plants were held in a moist chamber for 1½ to 3 days and then removed to the greenhouse. From 5 to 7 days after inoculation small, necrotic spots appeared on the inoculated leaflets and cotyledons. Uninoculated plants held under the same conditions remained healthy. Close examination, with high magnification, of the lower surface of the inoculated leaflets revealed many small, sunken areas where the mesophyll had collapsed. The central portion later became necrotic, ultimately enlarging to a maximum diameter of 2 mm., and was surrounded by a halo of chlorotic tissue (Fig. 1). Frequently the tips of immature leaflets were attacked, and in such cases the necrotic tissue was more extensive. Lesions on cotyledons were larger than on leaflets and were predominantly at the tip or near the margin. From one-fourth to one-third of the cotyledon was invaded before it abscissed.

Spores typical of *Colletotrichum phomoides* were observed when individual lesions were crushed and examined microscopically. In most instances only 2 to 3 spores were observed for each lesion examined, but in a few cases spores were abundant. Definite acervuli were not seen.

In one experiment at the Riverton laboratory 240 plants were inoculated. All became infected and the number of lesions varied from 4 to 198 per plant. Isolations were made from 30 leaflets 8 days after inoculation. The infected tissue was immersed in a 1–1000 solution of mercuric chloride for 15 minutes and washed in several changes of sterile water to remove surface contaminants. Platings were made on potato dextrose agar. *Colletotrichum phomoides* was recovered from 28 platings, while 2 remained sterile. Two

[1] Rolfs, F. M. Report of the horticulturist. Anthracnose. Florida Agr. Exp. Sta. Ann. Rept. 1905: 45–46. 1906.

[2] Giddings, N. G. Potato and tomato diseases. West Virginia Agr. Exp. Sta. Bul. 165: 19, 20. 1917.

of the recovered isolates were used to inoculate tomato seedlings and in both cases leaflet lesions developed similar to those described. In another isolation experiment 14 infected cotyledons, 20 infected leaflets with necrotic spots, and 20 leaflets with infected tips were surface sterilized and plated on potato dextrose agar. Twelve of the lesions from cotyledons, 18 of the leaflet spots, and 18 of the infected leaflet tips yielded *C. phomoides,* while the

FIG. 1. Tomato leaflets of the Rutgers variety infected with *Colletotrichum phomoides.* Photographed 9 days after inoculation. Small, circular, necrotic spots are surrounded by halo of chlorotic tissue. Marginal infections are larger, irregular, necrotic spots.

remaining 6 platings were sterile.—S. G. YOUNKIN, Research Department, Campbell Soup Co., Riverton, N. J., and A. W. DIMOCK, Cornell University, Ithaca, N. Y.

Intercellular Mycelium of Taphrina deformans in Peach Fruit.—A recent paper by Hildebrand[1] regarding the occurrence of *Taphrina deformans* (Berk.) Tul. on peach fruits has emphasized the paucity of direct evidence for the existence of the organism in such material. Hildebrand was unable to find any trace of the fungus in the maturing fruits that he examined, and, as far as the writers are aware, only three statements[2,3,4] of the actual pres-

[1] Hildebrand, E. M. Mature peach fruits affected by leaf curl. Phytopath. 34: 345–347. 1944.

[2] Clinton, G. P. Report of the botanist for 1913. Conn. Agr. Exp. Sta. Ann. Rept. 1914, Pt. I: 1–42. 1914.

[3] Arnaud, Gabriel, and Madeleine Arnaud. Traité de pathologie végétale. Tome I, Vol. 2, 995–1831. Paris, 1931.

[4] Rose, Dean H., D. F. Fisher, Charles Brooks and C. O. Bratley. Market diseases of fruits and vegetables: peaches, plums, cherries and other stone fruits. U. S. Dept. Agr. Misc. Publ. 228: 26 pp. 1937.

ence of the fungus exist in the many descriptions in the literature of peach fruits which, by their symptomatology and association, were believed to be infected with the leaf curl fungus. The evidence for the existence of *T. deformans* on the fruit, therefore, appears to be chiefly circumstantial. The possibility exists, however, that other factors such as viruses or insects may be involved in the expression of the previously described fruit symptoms—discolored, slightly raised lesions or warty protuberances. In this connection, a comparison of Blodgett's findings on peach wart[5] with some of the descriptions and illustrations of peach fruit presumed to be infected with *T. deformans* reveals a marked similarity in symptomatology between the two diseases. For this reason, examination for the presence of internal mycelium was made of various California collections of half-grown and nearly mature peach fruits with characteristic lesions or warty protuberances, or, in some cases, symptoms intermediate between these two types.

Fruits with discolored, slightly raised lesions similar to those illustrated by Smith[6] were sent to us from Davis, California, by Dr. E. E. Wilson in 1942. Freehand sections revealed a small amount of sub-epidermal mycelium, essentially similar in appearance to that found in infected leaves. In a personal communication, Dr. Wilson stated that he had actually found mature asci and ascospores on the surfaces of some of these peach fruits, an observation similar to those made by Clinton[2] and G. and M. Arnaud[3] on peaches and to that of Cunningham[7] on infected nectarine fruits. Preserved herbarium specimens from unknown localities in the State exhibiting, for the most part, slightly raised lesions were examined microscopically and yielded abundant intercellular, septate mycelium (Fig. 1, A, B, C, D) which was sometimes 2½ mm. below the epidermis. In addition, sub-cuticular mycelium (Fig. 1, E) and ascogenous cells were found. As noted by other investigators,[1,7] the trichomes were scarce or lacking on the surfaces of the slightly raised lesions.

No trace of the fungus was found through histological examination of fresh and preserved specimens from other California collections, most of which consisted of fruits with the warty type of symptom similar to that illustrated by Rose[4] and in which there appeared to be no reduction in the number of trichomes on the surface. Because of the readiness with which the mycelium was demonstrated in fruits with the other type of lesion, it appears doubtful whether these warty specimens were actually infected with the peach leaf curl fungus.

Since some of the fruits examined possessed symptoms that were intermediate between the warty type and the type with slightly raised lesions, it appears that no definite correlation is possible between macroscopic appearance of the fruit and the presence of the fungus. Thus, because of the possibility that factors other than the organism may be responsible for the effects

[5] Blodgett, Earle C. Peach wart. Phytopath. 33: 21–32. 1943.
[6] Smith, Ralph E. Diseases of fruits and nuts. Calif. Agr. Ext. Circ. 120. 1941.
[7] Cunningham, G. H. Leaf curl, bladder plum and cherry curl. Their appearance, cause and control. New Zealand Jour. Agr. 26: 85–97. 1923.

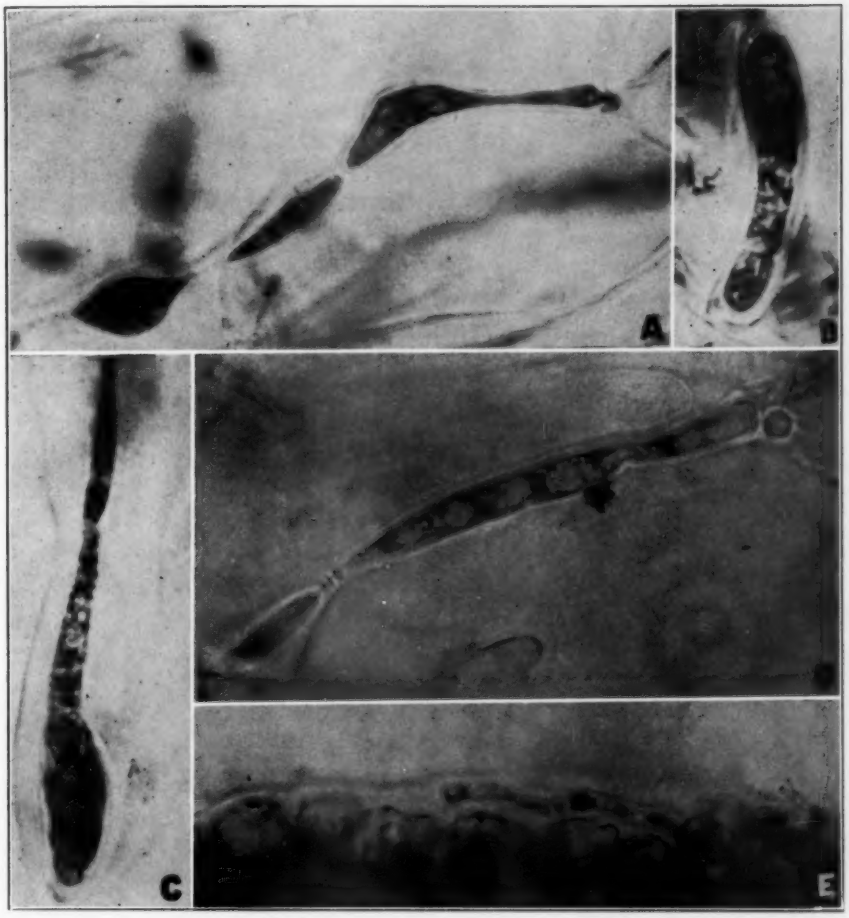

FIG. 1. Intercellular mycelium of *Taphrina deformans* in peach fruit. A, B, C, D. Sub-epidermal hyphal strands. E. Sub-cuticular hyphal strand. Stained with 0.1 per cent cotton blue in lactophenol. (A, D, E, × 850; B, C, × 1200).

observed, it is, in the writers' opinion, unwise to state with assurance that peach fruit is infected with *T. deformans* merely upon the basis of symptomatology.—CATHERINE ROBERTS and J. T. BARRETT, Division of Plant Pathology, University of California, Berkeley 4, California.

Hot-water Treatment for Control of Phytophthora Root Rot of Calla.—Root rot of calla (*Zantedeschia* sp.), caused by *Phytophthora richardiae* Buis., is the most common and destructive disease of this popular ornamental in eastern greenhouses. The damage results from extensive rotting of the roots which leads, in turn, to dying of the foliage and partial or complete failure of the flower crop. In most cases the pathogen penetrates very slightly into the rhizome tissue at the point of attachment of the roots and remains viable in such lesions for many months under the usual conditions

of handling. Such infected rhizomes thus serve to carry the pathogen from one season to the next within an individual greenhouse, and serve as the principal, if not the sole, means of disseminating the organism over long distances.

Fortunately, almost complete eradication of the pathogen can be effected by soaking infected rhizomes for 1 hour in a 1–50 formaldehyde solution,[1] in a 1–1000 mercuric chloride solution,[2] or in a suspension of 1 lb. of New Improved Ceresan plus 2 oz. Dreft in 50 gallons of water.[3] The treated rhizomes should be planted in soil and containers free of the pathogen.

The successful use of hot-water treatments in the control of other diseases caused by Pythiaceae (reviewed by Baker and Cummings[4]) suggested that such treatments might here be used in lieu of the chemical treatments for calla rhizome disinfection.

FIG. 1. Control of Phytophthora root rot of calla by hot-water treatment. Left, untreated checks; right, rhizomes soaked for 1 hour at 122° F. prior to planting. Both lots planted in sterilized soil. Photographed after 128 days.

Thirty dormant white calla rhizomes known to have been infected during the 1943 season were lined out on the laboratory table according to size. Every second rhizome was taken for treatment and the remainder were used as untreated controls. Because of the limited sample only one treatment was employed, a hot-water soak at 122° F. (50° C.) for one hour. On the basis of previous work[4] this was thought adequate to kill the fungus, and had been found in extensive trials[5] to be safe for dormant calla rhizomes. The treatment was made November 8, 1943, and both treated and untreated lots were planted in sterilized soil in sterilized pots 2 days later. The two lots were separated by a considerable distance, but were kept in the same greenhouse section. The pots were not in contact with the bench soil.

[1] Buisman, C. J. Root rots caused by Phycomycetes. Meded. Phytopath. Lab. "Willie Commelin Scholten" Baarn. **11**: 1–51. 1927.

[2] Tilford, P. E. Calla lily root rot and its control. Ohio Agr. Exp. Sta. Bimonthly Bull. 157: 138–140. 1932.

[3] Creager, D. B. Successful culture of callas requires prevention of diseases. Flor. Rev. 92 (2382): 11–12. 1943.

[4] Baker, K. F., and K. Cummings. Control of Pythium root rot of *Aloe variegata* by hot-water treatment. Phytopath. **33**: 736–738. 1943.

[5] Unpublished tests by Stephen Wilhelm, University of California, Los Angeles.

A few of the untreated plants had definite symptoms of root rot on January 20, and by February 28 all untreated plants were infected. On March 17 the foliage of all untreated plants was practically dead and the root systems were completely rotted, while neither root infection nor foliage symptoms could be detected in any plants of the treated series (Fig. 1). The experiment was then discontinued.

The perfect results obtained in this exploratory test with known infected material indicate that heat-treatment may safely be employed as an alternative to chemical treatment of infected rhizomes in the control of Phytophthora root rot of calla. There was no evidence of delay in either sprouting or blooming in this test comparable to that reported from chemical treatment.—A. W. DIMOCK, Cornell University, Ithaca, N. Y., and K. F. BAKER, University of California, Los Angeles, California.

Two New Records of Sphaceloma Diseases in the United States.—In July, 1943, diseased leaf specimens of *Aralia spinosa* L. from a nursery in northeastern Missouri were received from C. M. Tucker, of the University of Missouri, for identification of the fungus present. The severely diseased growth was affected by scab caused by *Sphaceloma araliae* Jenkins[1] (Fig. 1, A). On the advice of Dr. Tucker the entire planting of *Aralia* was removed from the nursery and destroyed by burning in order to avoid the spread of the disease from that source. Previously the scab had been known only from Maryland, where it was discovered at Edgewater, Anne Arundel County. The affected plants were in a wild plant garden where they had been transplanted from their native habitat. All of the diseased growth was removed and preserved as herbarium specimens. The disease did not recur. On his visits to the same county, that is, to Herald Harbor, which is about 10 miles from Edgewater, J. A. Stevenson has examined wild *Aralia spinosa* for scab, but none has been found. In 1941, using a culture isolated by the writer in 1934, Mr. Stevenson made a few inoculations on plants growing on his property at Herald Harbor. A limited number of lesions resulted on the inoculated foliage, which also was gathered for an herbarium specimen. As in the previous instance, no subsequent trace of the disease was found either on the inoculated or on nearby uninoculated plants.

Diseased willow leaves gathered in Skagit County, Wash., in August, 1943, by inspectors of the Bureau of Entomology and Plant Quarantine and referred to the writer for examination have been recognized to be affected by gray scab caused by *Sphaceloma murrayae* Jenkins and Grodsinsky (Fig. 1, B). (See Grodsinsky and Jenkins,[2] page 56, footnote 1.)

[1] Jenkins, A. E. New species of *Sphaceloma* on *Aralia* and *Mentha*. Jour. Wash. Acad. Sci. 27: 412–414. 1937.

[2] Grodsinsky, L., and A. E. Jenkins. *Sphaceloma murrayae* en diversas especies de *Salix*. Rev. Argentina Agron. No. 1, 10: 55–58. 1943 (issued March 15, 1943). The diagnosis of *S. murrayae*, just cited, is quoted from that appearing in the text of the article by Jenkins and Grodsinsky entitled "Sphaceloma on Willow in New Zealand" (Trans. Brit. Myc. Soc. Parts I and II, 26: 1–3. 1943. Issued April 8, 1943). Although the article in the "Transactions" was in press in advance of that in the "Revista," it was published slightly later as just indicated. For reasons of priority, therefore, the quoted diagnosis is the initial one.

The willow was identified by C. R. Ball as the Pacific Coast species, *Salix lasiandra* Benth. This is the first record of gray scab on this species of *Salix*, as well as the first from Washington State.

The hosts and geographic range of *Sphaceloma murrayae* as previously known[3] may be summarized as follows: *Salix babylonica* L., New Zealand; *S. fragilis* L., Rhode Island, New York, Virginia, and New Zealand; *S. fragilis* × *alba*, Virginia; *S. lasiolepis* Benth., California; *S. viminalis* L., Latvia, also on this species as cultivated in the Delta del Paraná, Argentina.

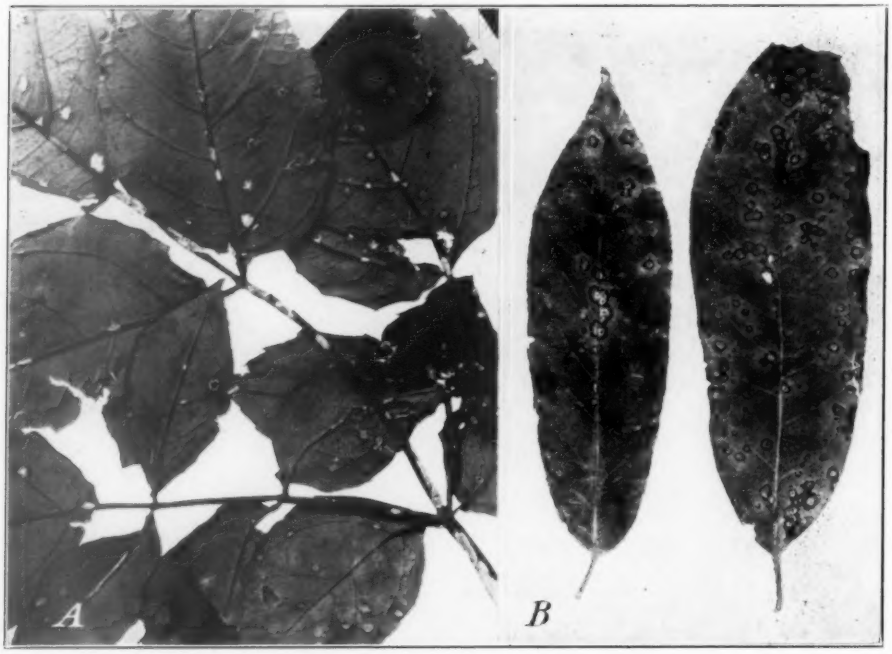

FIG. 1, A. Scab of *Aralia* on *Aralia spinosa*, northeastern Missouri, August, 1943. J. A. Denning. B. Gray scab of willow, on leaves of *Salix lasiandra*, Skagit County, Wash., August, 1943, gathered by inspectors of the U. S. Bureau of Entomology and Plant Quarantine. Photographs (×1) by R. L. Taylor.

Based on this distribution of *Sphaceloma murrayae*, Dr. Ball has furnished the following comment:

"It is interesting to consider the implications of three facts now apparent. 1. This fungus now is known to occur on five species of *Salix*, belonging to four Sections of the genus (Lucidae, Fragiles, Lasiolepes, and Viminales). Only the first two are closely related. 2. It has been collected on four continents, the countries represented being New Zealand, Latvia, Argentina, and the United States. In the last, it has been found in Rhode Island, New York and Virginia, in the East, and in California (S. W., Central, and N. W.) and in Washington, in the West. 3. Nearly all of the collection localities known to date are coastal and all are within the reach

[3] *Loc. cit.*, cf. articles cited in footnote 2.

of ocean influences. This may or may not be significant. These three facts, taken together seem to indicate that this fungus may occur still more widely than yet known, both as to host species and as to geographic range, especially coastal."—ANNA E. JENKINS, U. S. Plant Industry Station, Beltsville, Maryland.

Experimental Control of Orange Decays with Thiourea.—Stem-end rots, caused by *Diplodia natalensis* and *Phomopsis citri*, and green mold, due to *Penicillium digitatum*, of citrus fruits have long caused serious economic losses. In preliminary experiments between January and July, 1944, thiourea has been highly effective in preventing the development of these organisms on midseason and late varieties of oranges such as Pineapple, Temple, Valencia, and seedling types. Before its commercial use on citrus fruit can be recommended, however, it will be necessary to have more complete knowledge relative to the possible effect on public health.

Fruit used in this work was either clipped directly from the trees or obtained from packing houses. Usually about 40 fruits were used for each treatment within an experiment, a number deemed sufficient because of the negligible amount of decay in treated lots in contrast to the large amount in checks. In most cases the fruit was exposed to ethylene vapors 40 to 50 hours before experimental treatment, because such exposure favors the development of stem-end decay. This gassing treatment assured a high percentage of decayed fruit in the check lots and at the same time served as a severe test of the efficacy of thiourea. To afford maximum opportunity for decay, the fruit was stored at 70° F. for 3 weeks before final readings were taken. Hence, it should be emphasized that the percentage of decay was considerably higher than is ordinarily obtained under commercial conditions.

In-vitro tests with thiourea dissolved in cornmeal agar (Difco, 1.9 per cent) showed that *D. natalensis* grew at 0.1 per cent but not at 0.2 per cent concentration and that *P. citri* and *P. digitatum* grew at 0.01 per cent but not at 0.02 per cent concentration.

Concentrations of thiourea covering a range from 10 per cent to 0.1 per cent were tested in several experiments. Approximately 50 per cent control was obtained with 1 per cent concentration and over 90 per cent control was obtained with 4 per cent (Table 1).

The solubility of thiourea is 9.18 gm. per 100 cc. at 13° C. In 164 fruits (4 experiments) dipped in 10 per cent thiourea at 21° C. there was 2.5 per cent decay; in 179 check fruits there was 30.7 per cent decay. Thus the 10 per cent concentration did not give a significant increase in control over that obtained with 4 per cent thiourea (Table 1). A 2 per cent solution was only slightly less effective than the 4 per cent.

A 5 per cent solution, which is well within the solubility range of thiourea, was arbitrarily chosen for extended tests. In seven lots of treated fruit (225 fruits) 1.8 per cent decayed; in the seven non-treated lots (222 fruits) 38.2 per cent decayed (Table 1).

One of the questions considered was the possible value of a wetting agent in the dip. Vatsol O.T., 0.05 per cent, was used, but later tests indicated that it gave no improvement in control. Nevertheless the use of a wetting agent was continued to insure more complete coverage.

Tests were made to determine the extent to which the effectiveness of thiourea is altered by washing or brushing the fruit subsequent to dipping. A greater degree of control was secured when the thiourea was dried on the fruit before washing. When the fruit dipped in a 5 per cent solution of thiourea was dried before washing there was no decay; when washed while still wet, there was 19 per cent decay, compared to 44.6 per cent decay in the check lots. Similar experiments were made in which fruits were dried by running them over 35 feet of revolving horsehair brushes. When the treated fruits were brushed while still wet only partial control was obtained (10 per

TABLE 1.—*Effect of various concentrations of thiourea on control of stem-end and green mold rots*

Concentration of thiourea	No. fruits			Fruits rotted	
	Total	With stem-end rot	With green mold rot		
Per cent				*No.*	*Per cent*
10	164	0	4	4	2.5
Check	179	38	17	55	30.7
5	225	3	1	4	1.8
Check	222	60	25	85	38.2
4	117	0	3	3	2.6
2	184	6	2	8	4.3
1	182	25	4	28	15.9
0.5	186	42	10	52	27.9
0.25	146	38	9	47	32.2
Check	184	61	5	66	35.8
0.10	147	45	10	55	37.4
Check	147	45	3	48	32.6

cent decay in the treated lots; 43.3 per cent decay in non-treated lots). Unfortunately, there were no experiments in which treated fruits were air dried before brushing. But in a single experiment with 80 fruits, excellent control was secured when the fruits were treated and air dried, after brushing. There was no decay in the treated lot; 49 per cent decay in the non-treated lot.

To prevent wilting of the fruit during storage several lots were waxed after the thiourea treatment by dipping them in a proprietary wax emulsion. It seemed desirable to test the effectiveness of thiourea combined with the emulsion. In two experiments with 252 Valencia fruits gassed 40 and 42 hours, there was no decrease in control when thiourea was applied in the water phase of the wax emulsion. The 5 per cent thiourea treatment resulted in 2.4 per cent decay and the 5 per cent thiourea plus wax emulsion, 1.2 per cent decay; and 34.5 per cent of the non-treated fruit decayed during 18 days of storage.

Several investigators have reported the causal organisms of stem-end rots

present in the "buttons" (calyx, receptacle, and part of pedicel) at picking time. After frequently observing the marked browning effect of thiourea on the "buttons," an experiment with 100 non-gassed fruits was made. Thiourea (5 per cent) caused browning of 48 per cent of the green buttons, while natural browning occurred on 16 per cent of the non-treated checks in 13 days. Ethylene likewise causes button browning but at the same time increases stem-end decay. It is obvious that browning *per se* is not a factor in stem-end rot control and that the decay-inhibiting effect of thiourea cannot be ascribed solely to its phytocidal effect on the "button" tissues. The marked increase in browning produced by thiourea is contrary to what might be anticipated from an anti-oxidant.

Some evidence that the linked sulfur of thiourea is important to its fungicidal activity was obtained from an experiment in which 69 gassed Valencia oranges were (a) dipped in 5 per cent thiourea, (b) dipped in 5 per cent urea, and (c) were not treated. At the end of 16 days there was no decay in (a), 56 per cent decay in (b), and 47 per cent decay in (c). Additional evidence that the linked sulfur is important was obtained from *in-vitro* tests. Media containing thiourea and media containing urea, in which the concentration of the $=C(NH_2)_2$ fraction was the same over a wide range of dilutions, inhibited growth of these fungi only when the linked sulfur atom of thiourea was present. In limited tests with several inorganic sulfur compounds no significant decrease in decay was noted.

Thiourea thus gave definite experimental control of stem-end rot and green mold decays of oranges. Although the data on gassed and on non-gassed fruit have not been segregated in this report, it is highly significant that thiourea is effective in controlling these decays even when applied after the fruit has been exposed to ethylene.—J. F. L. CHILDS, and E. A. SIEGLER, Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, United States Department of Agriculture.

A Canker and Some Decay Fungi on Mimosa.—The mimosa tree (*Albizzia julibrissin* Durazz.) occurs abundantly in the southern part of the United States. Although a native of Asia, it is so well suited to the growing conditions encountered in much of our South that it has escaped from cultivation and now occurs in the wild at many places. This is the hardiest of the *Albizzia* species but it cannot be grown very far north of Washington, D. C. Even at Washington, it is so near the extreme northern limits of its range that it sometimes suffers from winter injury; and twigs and branches killed during the winter are common.

A recent examination of a number of mimosa trees in the District of Columbia disclosed numerous cankers (Fig. 1) on otherwise healthy branches and recalled an earlier collection made in 1935. Obviously many of the cankers, although present a number of years, have not caused progressive dying, and have resulted in relatively little damage. Smaller twigs

and branches were in some instances girdled but branches over an inch in diameter were seldom so severely affected. In each instance the canker had developed around the base of a smaller dead twig, indicating that the causal organism may be able to attack healthy wood only when it has been previously established in an adjoining dead portion. The fungus present in all cases was the ubiquitous *Nectria cinnabarina* Fr., for the most part in its imperfect stage (*Tubercularia vulgaris* Fr.). This fungus was isolated and obtained in culture from the dead wood of the cankered areas.

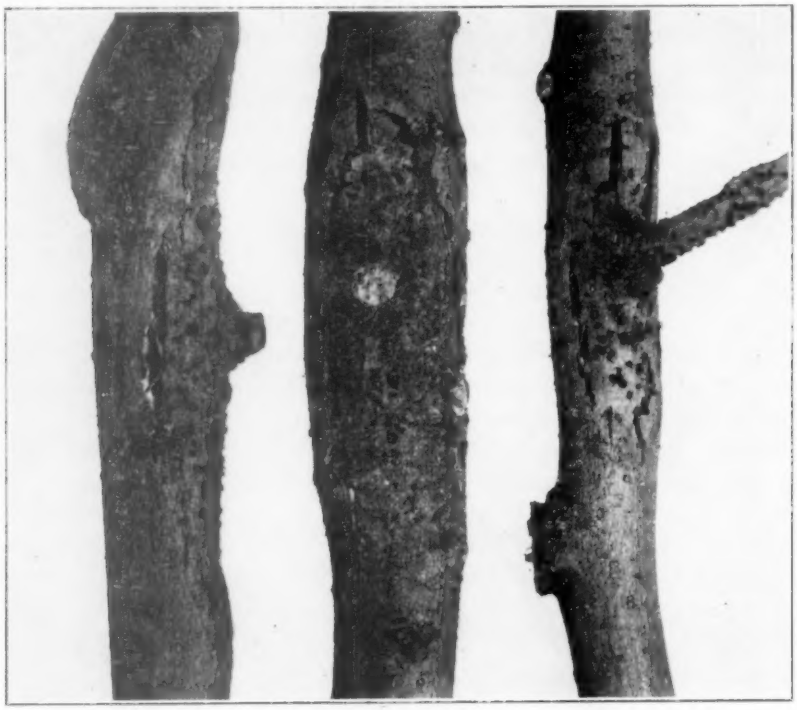

FIG. 1. Cankers on mimosa, each with a dead twig near its center and abundant fruiting of the *Tubercularia* stage of *Nectria cinnabarina* Fr. (Natural size.)

Nectria cinnabarina has been reported from a very wide range of woody plants, and from every continent in the world. To a considerable extent it is reported as a saprophyte on dead twigs and branches only, but in some instances, as in the present case, as a wound parasite associated with cankers following drought or winter injury. It has been studied particularly as an organism associated with senescence and decline of apple and other fruit trees and shrubs. Only two previous published reports of the fungus on *Albizzia* have been found: one by Dr. Alma Waterman (Plant Disease Reporter **23**: 85, 1939) from North Carolina and one by D. P. Limber (Plant Disease Reporter **16**: 138, 1932) in the *Tubercularia* stage from Virginia. Specimens have also been collected in South Carolina and Maryland in 1938 by members of the Division of Forest Pathology.

Although the *Nectria* was the predominant fungus noted, other fungi were present on dead and dying branches and doubtless were contributing to the decline of the host trees. Five species in particular were wood-decaying fungi: *Polyporus versicolor* Fr., *P. hirsutus* Fr., *P. tulipiferus* (Schw.) Overh., *Stereum albobadium* (Schw.) Fr. and *Schizophyllum commune* Fr. *Eutypella stellulata* (Fr.) Sacc. was also present in abundance on dead twigs and branches. All are common on other woody hosts, but do not appear to have been reported previously on *Albizzia*.—MARVIN E. FOWLER and JOHN A. STEVENSON, Plant Industry Station, U. S. Department of Agriculture, Beltsville, Maryland.

BOOK REVIEWS

BAWDEN, F. C. *Plant Viruses and Virus Diseases.* Second Edition. XI + 294 pp., 48 illus. Chronica Botanica Co., Waltham, Mass. 1943. Price $4.75.

The author deals almost entirely with those viruses that have received the most intensive studies, and, as in the first edition, emphasis is placed on the biochemical and physical studies. The literature lists contain approximately 640 citations, about 150 more than the first edition. Two cases are noted in which papers are mentioned in the text but not listed with the references, *i.e.*, on page 100, McKinney (1937) and page 140, Mulvania (1926). The book is well illustrated and the tables are understandable.

In Chapters 1 and 6 some of the author's views show a noticeable bias. His interpretations of plant-virus and plant-disease phenomena seem to be unduly influenced by traditional views and facts pertaining to the vertebrates. This is especially noticeable in connection with some of his views relating to virulence, vaccination, and acquired immunity. No mention is made of the sparing effect or interference manifested by some of the viruses of the vertebrates. In a book of this scope, this subject merits full discussion in relation to the unilateral interference or antagonism (''acquired immunity'') which occurs between certain plant viruses. On page 97 the author seems to ignore completely the fact that the term *mutation* was in use long before it was employed in connection with the organisms that reproduce sexually. Attempts to preempt this term for the sexually-reproduced organisms are little short of pure sophistry.

Chapter 2, on external symptoms, strikes one as being rather brief for a book of this character. Chapter 3, on the internal symptoms, is brief, but the author has given a good résumé of the subject. Chapter 4, on virus transmission, is very brief, and there is a tendency to generalize—perhaps too much—from the studies on the tobacco mosaic virus. In table 3 it is not clear why the author regards the two species of true bugs, *Lygus pratensis* and *Piesma quadrata*, as leafhoppers.

Chapter 5 deals with viruses in relation to insect vectors. The author is critical of some of the interpretations put forward for the increase of virus in the leafhoppers, but it appears to the reviewer that the criticisms are essentially constructive. With reference to the author's discussion of Black's data on page 80, his statement, ''it is noticeable that the number of successful inoculations is usually greater if the extracts of macerated insects is diluted 1/1000 than if diluted 1/100 or 1/10,'' seems not to be misleading as claimed by Kunkel in his review (Science **99**: 450, 1944). Granting that the infective differences shown between the dilutions of 1/1000 and 1/100 are very small and probably not statistically significant, the fact remains that the complete data do give a strong indication that the virus was more infective in the insects when it was diluted. Bawden could have given more consideration to those of Black's data which do indicate increase of virus in the inoculated insects in the final tests. However, if there is a true decline in virus concentration in the inoculated insects after approximately the 12th to 16th day from inoculation, as thought by Black, it appears from his control-insect data that the insects which fed all their lives on infected plants contained an additional reservoir of virus that had been accumulating directly from the infected plants as indicated by Bawden. It appears that there is more to this problem than can be brought out in a brief discussion. Excessive condensing of subject matter in the book doubtless explains some of the author's statements which give rise to controversy. On page 76 the statement, ''in published work there is no indication that vectors can ever infect healthy plants immediately after leaving infected ones,'' is a case in point. In the the interpretations being discussed time is a very important element, the word ''immediately'' therefore is not appropriate, for ''immediately'' is without time, and obviously time is required for all insect vectors to settle and begin feeding in a new location.

In Chapter 7 the author gives a very good résumé of serological methods, and of the results obtained with reference to plant viruses. He favors the use of the serological properties in virus classification, and in Chapter 14, on the classification of viruses, he gives rather serious consideration to the idea that these properties should be accorded essentially a generic rank.

Chapters 8 to 12 inclusive deal with the methods of virus purification, chemical and physical properties. The author has dealt with these fields very effectively. However, on page 140 he indicates that Mulvania in 1926 was the first to show ''that tobacco mosaic virus could be precipitated by protein precipitants and resuspended without losing its infectivity.'' Mulvania's paper is not cited in the list of literature, but it seems probable that the paper referred to is the one published in Phytopathology **16**, p. 583. This paper certainly does not support Bawden's statement. Mulvania used HCl, NaCl, NaOH, and glucose to determine their effects on the dializability of the virus through different grades of membranes, and he speculated on the possible protein nature of the virus, but he did

not mention precipitation or resuspension of the virus. Vinson and Petre, mentioned only casually by Bawden, were the first to record this and to show that the virus can be studied effectively by chemical methods. Stanley was the first to obtain virus in reasonably pure form and in large quantities for intensive study, and Bawden and Pirie were the first to show that the virus is a paracrystalin nucleoprotein and not a crystalin globulin. Furthermore, Bawden and his co-workers made important contributions in connection with the ultra purification of the virus.

Chapter 13, on the physiology of virus-diseased plants, is rather brief. While the literature on this subject is not very extensive, a more extensive résumé would have been welcomed. Chapter 14, on virus classification, covers the important proposals advanced by other investigators, and the author expresses his own views on the subject quite frankly. He seems to favor a system of scientific naming rather than a system of numbering, and he looks with decided disfavor on host reactions as a basis for classification. Chapter 15, on the control of virus diseases, is very introductory. Here the author seems to be somewhat out of his field. Chapter 16, on the origin and multiplication of viruses, is largely speculative. The author does not take the position that the viruses are definitely nonliving chemical entities. This is especially significant when it is considered that he has been actively engaged in advancing the study of viruses by physical and chemical methods.

This book contains much valuable information, and it should stimulate a critical viewpoint among virologists; for this the author deserves high commendation.—H. H. MCKINNEY, U. S. Bureau of Plant Industry Station, Beltsville, Maryland.

BAXTER, D. W. *Pathology in Forest Practice.* 618 pp., 232 figs. John Wiley and Sons, New York. 1943. Price, $5.50.

The problems of pathology in forest practice are discussed in the aspect in which they present themselves to the forester in the nursery, the plantation, the forest, the park, and in industry. Consequently little attention is paid to the small details of taxonomy, mycology, and etiology. Stress is placed on a working knowledge of the conditions which favor the development of various diseases and on the practical measures which can be taken to reduce their incidence or severity.

After a discussion of the nature of a plant disease, the causal fungi are briefly described. Emphasis is placed on the higher Basidiomycetes because they include the principal wood-destroying fungi. The various methods are given of determining loss and of making an appraisal of damage. Particular attention is given to critical relationships, such as (1) those of site and of cultural practice to the incidence of disease in the nursery, in plantations, and in the mature forest, (2) those of fungi and of certain other plant pests to disease in the mature forest, and (3) those of site and of care given shade and park trees to disease. Forest products are considered from the standpoints of decays, discoloration, stains, molds, and other defects.

The publication of this book is particularly timely, because during the war devastating inroads are being made on the nation's timber supply and because after the war many returned soldiers and war workers will find employment in extensive reforestation. Professor Baxter has assembled the information for those planning and directing the forestry programs so they can avoid costly and time-consuming errors. He has attacked a difficult problem in a masterful and authoritative way.

The printing is excellent both of the text and of 232 illustrations. A large number of literature citations appear at the end of each chapter. There is an excellent index.

Among technical books on forestry this one will appeal to practical men. It is considered indispensable for anyone who seeks to understand the conditions which favor tree damage by disease and with this information either to provide protection or to reduce the severity of the injury.—A. J. RIKER, University of Wisconsin, Madison, Wisconsin.

REPORT OF THE FIRST ANNUAL MEETING OF THE POTOMAC DIVISION OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY, BUREAU OF PLANT INDUSTRY STATION, BELTSVILLE, MARYLAND, FEBRUARY 23 AND 24, 1944

The first annual meeting of the Potomac Division of the American Phytopathological Society was held at the Bureau of Plant Industry Station, Beltsville, Maryland, on February 23 and 24, 1944.

Research papers were presented on the mornings of February 23 and 24; the afternoon of February 23 was spent in discussing quarantine protection problems under the leadership of Dr. W. A. McCubbin; and a business meeting on the afternoon of February 24 was followed by a discussion on Wartime Work in Plant Pathology led by Dr. R. J. Haskell.

The following officers were elected: H. B. Humphrey, President; W. J. Zaumeier, Vice-President; T. F. Manns, Secretary and Treasurer; and H. P. Barss, Representative on the Council.

ABSTRACTS OF PAPERS PRESENTED AT THE FIRST ANNUAL MEETING OF THE POTOMAC DIVISION

Some Virus Diseases of Alliums. PHILIP BRIERLEY and FLOYD F. SMITH. Onion yellow dwarf is transmissible to and from wild garlic (*Allium vineale* L.). Garlic mosaic from late or Italian garlic received from Oregon is transmissible to onion and to *A. vineale* by sap and by *Myzus persicae* Sulz. Symptoms differ from those of yellow dwarf in both suscepts and the efficiency of transfer to onion is lower. Garlic mosaic in Spanish garlic has failed to transfer to healthy Spanish garlic, to *A. vineale*, or to onion in parallel tests by *M. persicae*. A mosaic of the ornamental *A. neopolitanum* Cyr. is transmissible to seedlings of this species by *M. persicae*, but not to onion in parallel tests. Aster yellows transferred from lettuce by *Macrosteles divisus* Uhl. induced yellowing and dwarfing in 3 of 10 Ebenezer onions in one trial, reproducing symptoms occasionally observed in field-grown onions that have been replanted in greenhouses for seed production.

Tobacco Disease Control by Rotation. E. E. CLAYTON. Crop rotation experiments on the control of soil-borne diseases affecting flue-cured tobacco were conducted from 1926 to date. The occurrence of the stem-rot and sore-shin diseases of tobacco, caused by *Sclerotium rolfsii* and *Rhizoctonia solani*, was not influenced by the cropping system. Continuous culture of sclerotium-susceptible crops did not increase tobacco stem-rot losses, nor did the use of immune crops reduce these losses. The occurrence of root knot and bacterial wilt caused by *Heterodera marioni* and *Bacterium solanacearum* was much affected by rotation practices. The organisms did not react the same. Root knot was effectively controlled by bare fallowing, while bacterial wilt was not. Again, once a field area was infected with wilt, successive crops of tobacco were uniformly and severely affected, while with root knot successive crops of tobacco planted on the same area might range from severely to slightly diseased. Effective disease control by rotation depended on the use of immune and not resistant crops. Over a period of years the results secured with resistant rotation crops were not superior to those obtained with susceptible. Fusarium wilt of tobacco, caused by *Fusarium oxysporum* var. *nicotianae*, appeared only where tobacco was grown in rotation with sweet potatoes. The time required at different locations varied from 2 to over 10 years.

Advances in Sugar-beet Seed Treatment. G. H. COONS and J. E. KOTILA. Improved stands have usually resulted from treatment of sugar-beet seed balls with fungicidal dusts prior to planting. Finely ground mercuric chloride (in equal parts with copper carbonate and urea), mercuric iodide, 2 per cent Ceresan, New Improved Ceresan, and other organic mercury compounds were superior to copper carbonate or cuprous oxide for seed treatment. Two per cent Ceresan used in equal parts with copper carbonate frequently showed more effective and lasting protection against damping off than 2 per cent Ceresan alone. Because of several factors, seed treatment for sugar beets, however, did not gain general adoption. Use of sheared sugar-beet seed in 1943 as sparse seedlings of the prevailingly single-germ seed units has revived interest in seed treatment because complete stands uniformly, but sparsely, distributed in the row are essential for mechanized thinning. Treatments effective with whole seed have been equally effective with sheared seed. Arasan and Spergon, used as dusts, also are effective for treating sheared seed, the latter

requiring heavier dosage than the usual recommendations. To obtain precision in planting sheared seed, pelleting of the seed is under trial. Standard pill-making techniques are used to coat the sheared seed in order to obtain a heavier seed unit, more uniform in size. Fungicidal treatments can be employed and nutrients and other substances can be added to the coating material, thereby increasing control of damping off.

The Response of Cantaloup-Cucumber Grafts to Inoculation with Fusarium bulbigenum var. niveum f. 2. C. E. COX. Cucumbers are not susceptible to the cantaloup wilt disease caused by *Fusarium bulbigenum* var. *niveum* f. 2. Grafted plants were used in order to determine whether, (a) cantaloup and cucumber plants growing on roots of the opposite species would respond differently to inoculation with this vascular parasite than plants on their own root systems, and also, (b) whether the shoots of one species would affect the susceptibility of roots of the other species. The following 4 types of grafted plants were prepared in the greenhouse: cantaloup scions on cucumber stocks, cucumber scions on cantaloup stocks, cantaloup scions on cantaloup stocks, and cucumber scions on cucumber stocks. The plants were grown in sand culture and watered with a nutrient solution. The roots of each plant were inoculated with 10 cc. of a macerated fungus mat from a liquid culture. The cantaloup-on-cantaloup grafts developed the symptom sequence characteristic of ungrafted plants. None of the cucumber-on-cucumber plants developed symptoms of the disease. In the plants composed of a cucumber scion on a cantaloup stock the stock developed typical symptoms and the scions withered and died. Cantaloup scions on cucumber stocks remained healthy. The extent of invasion of the parasite into the grafted plants was determined by culturing from various points both above and below the graft unions. The scion did not affect the susceptibility of the root stock to invasion by this fungus and the relative susceptibility of the scions was not altered by the susceptibility of the root stock.

Phytophthora Fragariae Hickman and Methods of Testing Strawberry Plants for Resistance. J. B. DEMAREE and W. F. JEFFERS. Testing strawberry plants for resistance to red stele has formerly been done in naturally infested soil. In order to improve this method a new technic for determining resistance by artificial inoculation was developed. The red stele organism is multiplied on Petri plates of lima-bean agar and the cultures are cut in small pieces and placed in water. Here zoospores are produced which are drained off and used as inoculum. Plants are grown in 2-inch pots and when sufficient new roots have grown are placed in a cool greenhouse. The plants are knocked from the pots, the bottom hole of the pot is plugged and 5 cc. of the zoospore suspension is added and the plant replaced. Susceptible plants show typical red stele symptoms in 2–3 weeks. Plants which do not become infected are reinoculated and if still resistant are set in naturally infested soil for fruiting tests.

The Fungicidal and Phytotoxic Behavior of Some Selected Organic and Inorganic Copper Compounds as Related to Weathering. M. C. GOLDSWORTHY. Copper malate, copper maleate, copper oxalate, copper citrate, copper tartrate, copper sodium polyphosphate, copper silicate, cupric oxide, cuprous oxide, and basic copper sulphate were tested. When the residues were subjected to weathering for 2 weeks, during which the rainfall was scanty, the compounds were lethal to the conidia of *Sclerotinia fructicola*, generally in accordance with the initial ionic copper content of the residues. Initial solubility was not related to fungicidal effectiveness. In no case were the conidia capable of changing the initial solubility or availability. In a few cases only, changes in solubility or availability accompanied onset of rainfall. In tests to determine the phytotoxic effect on Elberta peach leaves, those compounds that were initially soluble (though not fungicidal) exerted phytotoxic effects regardless of onset of rainfall. These compounds, as well as those not causing injury before onset of rainfall, became much more injurious when precipitation occurred. This indicated that environment played a considerable part in solubilizing the copper compounds so as to cause or increase phytotoxicity, but had little effect on their fungicidal behavior.

Virus Disease on the California Wonder Pepper in Delaware in 1943. J. W. HEUBERGER. Plants used were produced from two seed sources in plant houses used exclusively for peppers. In the field, those from one seed source averaged 54 per cent mosaic, whereas those from the other averaged less than 1 per cent mosaic. Since plants were set at the same time and fields were adjacent in many instances, chances for natural infection were equal. As no mosaic was reported in the plant houses, the striking difference in percentage infection may indicate a differential susceptibility to mosaic infection in the field. A new virus-like disease was also present, characterized by a continuous yellow discoloration beginning at the petiole and extending toward the leaf apex. These infected leaves readily fall from the plant. Fruits present at time of infection later have a yellow discoloration, often accompanied by pitting. Fruits formed after infection do

not develop. Plants from the same seed source with high mosaic infection likewise had high susceptibility to this tentatively named "Yellow Leaf" trouble. Culturing from all parts of infected plants yielded no causal organism. Preliminary transmission studies indicated that this trouble may be virus induced.

Oedema of Cultivated Violet Identified as Scab. ANNA E. JENKINS. "Oedema" or "wart" of sweet violet was first reported by B. T. Galloway in his "Commercial Violet Culture" (1889), which embodies the experience of Dr. Galloway and P. H. Dorsett in growing violets under glass at Garrett Park, Md. Recent examination of a specimen of the so-called oedema collected at Garrett Park by Galloway makes it seem certain that this actually represents what is now known as scab of violet caused by *Sphaceloma violae* Jenkins. Leaf symptoms are in general agreement with those of various specimens of the scab now available for comparison. Petiole lesions also correspond to those of violet scab, and scrapings from them show hyphal and conidial development of the *Sphaceloma*. The historic Galloway specimen is in the Mycological Collections of the Bureau of Plant Industry, Soils and Agricultural Engineering, where it was formerly entered in the "Physiological Collection." At the time when the "oedema" was giving trouble at Garrett Park, violet growers on the Hudson were contending with it, as the writer has been informed by a veteran violet grower. He has stated, also, as Galloway indicated, that conditions favoring the disease are poor ventilation, too much moisture, and cold. As previous reports show, the disease has been more or less widespread on native wild violets in the District of Columbia area; in 1935 it was found in great abundance on a wild violet growing extensively in a glade in Rock Creek Park, Maryland, *i.e.*, in the same stream valleys as Garrett Park. Galloway's account of "oedema," together with the specimen from Garrett Park, may now be construed as the first record of the *Sphaceloma* disease of violet.

An Undescribed Leaf Spot of Tift Sudan Grass. C. L. LEFABVRE. This leaf spot was first found in 1939 by Dr. G. W. Burton in a greenhouse at Tifton, Georgia, on plants of common Sudan × Leoti sorghum (now Tift sudan grass). Lesions on the leaves sent to the author varied considerably, the smaller ones being 0.5 to 1.0 mm. in each direction, while the larger ones measured up to 20 mm. in length and 2–6 mm. in width. The lesions were round to elliptical, the long axis parallel to the leaf veins, and oftentimes the spots seemed to be limited in their spread by the veins. Many of the lesions had coalesced so that comparatively large portions of the leaves were affected. The most characteristic features of the lesions, however, were the alternate light tan and darker, more narrow bands of tissue, resulting in a ringed or zonate appearance. Lesions were usually surrounded by a narrow reddish-brown border setting off the affected area from the healthy tissue. Symptoms on Tift sudan are strikingly similar to lesions on corn caused by *Helminthosporium maydis*. A species of *Helminthosporium* was readily isolated from the lesions on Tift sudan and when compared with *H. maydis* from corn, the former had spores averaging 61 μ and the latter 80 μ long. No difference in width was observed but there was a striking difference in the number of septa in the spores of the two fungi. Spores of the *Helminthosporium* from Tift sudan averaged 4.75 septa, while those of *H. maydis* from corn averaged 7.7 septa. Since these differences were found in the spores taken either directly from the hosts or from cultures, it appears the fungus from Tift sudan is not *H. maydis* but an undescribed species of *Helminthosporium*. From inoculation trials this fungus appears to be restricted to sudan grass.

Fungicides for Controlling Bulb Scale Rot and as Carriers for Growth Substances in the Propagation of Easter Lilies. W. D. MCCLELLAN and N. W. STUART. Bulb scales from some Easter lily clons are very susceptible to a rot caused by *Fusarium oxysporum* f. *lilii* Imle. Effective protectants are needed in scale propagation. The following materials have been tested in the greenhouses at the Plant Industry Station, Beltsville, Maryland: New Improved Ceresan (5 per cent ethyl mercuric phosphate), Spergon (98 per cent tetrachloro-para-benzoquinone), Arasan (50 per cent tetramethyl thiuram-disulphide), Fermate (ferric dimethyldithio-carbamate), Zincate (zinc dimethyldithio-carbamate), and formaldehyde. Best results were obtained with Arasan. Spergon and New Improved Ceresan also afforded good protection. Formaldehyde injured the scales. Fermate and Zincate were relatively ineffective. There was greater bulblet production and root and shoot development from the scales treated with Arasan than from those treated with New Improved Ceresan, Spergon, or formaldehyde. New Improved Ceresan inhibited bulblet formation and root development, but when the Arasan treatment followed Ceresan this inhibition was overcome. There were 300 scales in each treatment. The total bulblet production from those scales treated with Ceresan was 239; with Arasan, 442; the Ceresan-Arasan combination, 494; and from the untreated checks, 273. This stimulation did not occur when Spergon was used with the Ceresan treatment. When

Arasan or Spergon was substituted for talc as a carrier for the growth substances, naphthaleneacetic acid and indolebutyric acid, bulblet yields were greatly increased. The highest bulblet yields occurred when Arasan was used as the carrier for naphthaleneacetic acid. Scales treated with this combination produced 503 bulblets compared with 337 for naphthaleneacetic acid combination, 410 for Spergon alone, and 273 for the untreated checks.

Effect of Extracts from Cultures of Fusarium eumartii on Different Varieties of Potatoes. M. A. PETTY. Hot tap water (70° C.) was poured into 90-day cultures of *Fusarium eumartii* growing on sterilized whole oats. After 2 hours this aqueous extract was decanted. Half of this extract was steam-sterilized at 15 lb. pressure for 30 min. Excised vines of greenhouse-grown Irish Cobbler, Kasota, and President potatoes were placed in tap water (check), 50 per cent non-sterile extract in water, undiluted non-sterile extract, 50 per cent sterile extract in water, and undiluted sterile extract. Irish Cobbler vines in unsterilized extracts were noticeably wilted in 4 hours, while those of Kasota and President were unaffected. In 24 hours, only the few terminal leaves of Cobbler tops in the 50 per cent sterile extract were unaffected; all other leaves of plants in the extracts had completely collapsed, excepting the checks, which were erect. There was only a slight interveinal wilting and browning of the lower one or two leaves of the Kasota and President varieties in the unsterilized extracts, otherwise they were unaffected. There was no wilting of either of these last two varieties until after 36 hours: however, by this time there was considerable bacterial activity in the solutions. Excised tops of Rutgers and Beefsteak varieties of tomatoes were not affected by the extract.

Evidence of Hybridization between Physiologic Races of Ustilago hordei in Passage through Host. V. F. TAPKE. Seed of Odessa (C.I. 934) barley, highly susceptible to *Ustilago hordei* races 3 and 6, was inoculated with a mixture of chlamydospores of those two races in equal proportion. The content of physiologic races in 25 of the resulting smutted heads then was determined by inoculating differential hosts. Fifteen of the smutted heads contained only race 3, one contained only race 6, seven contained a mechanical mixture of both races, and two contained a different race capable of attacking varieties immune from race 3 or 6. This race appears to have resulted from hybridization. Further study has shown that the synthetic race is not a new one but produces reactions of the differential barley varieties similar to that of a naturally occurring race. A few of the further selections of the hybrid in later generations lost the capacity to attack varieties immune from race 3 or 6 but most of them have maintained it.

Tobacco Anthracnose, a New Tobacco Plant Bed and Field Disease. E. A. WALKER and SARAH W. MCINROY. Tobacco anthracnose was observed as a leaf spot in commercial plant beds on May 24, 1941, near Waldorf, Maryland. The following year it was very destructive to tobacco plants in poorly drained soils. Diseased plants placed in fields continued to develop symptoms under wet conditions. In the plant bed anthracnose was observed as a leaf spot, seedling blight, leaf midrib and petiole and plant stem cankers. In the field these symptoms extended as the plant grew and cankers were found on the branches of the flower head and on the seed pods. Anthracnose could be easily spread by means of contaminated seed. A *Colletotrichum* sp. was consistently isolated from tobacco leaf spots and stem cankers and its pathogenicity for tobacco established. A *Gleosporium* sp. was also obtained from diseased tobacco plants and from sectors of *Colletotrichum* sp. in culture. Spiral growth was observed with some strains of both organisms on some culture media.

Studies on the Virus of Brome-grass Mosaic. H. H. MCKINNEY. The end-point of virus activity in native extract is 10 min. when heated near 78.5° C.; is near 10^{-5} when diluted with water; in dry leaf tissue at laboratory temperatures the end-point is beyond 306 days. Grass hosts include wheat, rye, barley, oats, sorghum, Johnson grass, sudan grass, teosinte, corn, and other annual and perennial grasses in 9 of the grass tribes. In corn seedlings (2–3-leaf stage), at high growing temperatures, the first signs appear 36 to 40 hours after inoculation. The reactions consist of chlorosis, necrosis, and death of the host. In other grass hosts, the reactions range from carriers showing no visible symptoms, through mild to severe chlorotic mottling and streaking. Natural resistance increases rapidly as host plants age. The first leaves of garden bean var. Scotia develop small local necrotic lesions, the cotyledons of cucumber var. Early White Spine and the leaves of Samsun tobacco develop local faintly chlorotic spots and there is local increase of virus in each host. Experimental inoculations were accomplished by the carborundum wiping method.

SUMMARY OF THE PAPERS PRESENTED AT THE PLANT QUARANTINE SESSION

Introductory Review. W. A. MCCUBBIN. It is encouraging that in its first meeting the Potomac Division should include a session on plant quarantine problems, thus recognizing understandingly that pathologists as a national organization have a responsibility in the field of foreign disease exclusion as well as toward national welfare and progress in teaching, research, and extension. This helpful interest is welcomed by the quarantine administration and is needed now, not only in the solution of immediate and postwar quarantine problems, but in the broader field of future national planning which should aim to establish the best permanent barriers against foreign disease introduction that science can devise, public opinion will support, and administration can maintain. Pathologists can contribute vitally to the future of our national plant quarantine system, especially in assuring that its various features rest on a sound biological basis. The national system of protection against foreign disease introduction may be considered as logically comprising four lines of defense,—the effort to secure extensive information on foreign pathogens as a requisite to effective understanding and planning; adequate control over the importation of pest-carrying materials in all its phases; domestic survey arrangements which would promptly disclose unfortunate disease introductions; and internal organization to deal with such introductions by eradication, by domestic state or federal quarantine action, or by control measures. Such a protective scheme contains many perplexing elements deserving of careful and earnest study by pathologists. In addition to the four chosen for discussion at this session, the foreign plant quarantine relations of the following merit much thoughtful attention,—nematodes; bacterial diseases; seed-borne diseases; foreign certifications; the importation of pathogen cultures; the value and limitations of inspection procedures; treatment possibilities; and the significance of races and strains of pathogens in the quarantine field. It is hoped that these and other aspects of the subject will come up for practical and profitable consideration in due course.

Quarantine Protection for Basic National Crops. HOWARD P. BARSS. Every enlightened government is impelled to include among its essential functions quarantine activities that aim to keep out of the country destructive plant diseases and pests which do not yet occur there. This is an integral part of the job of protecting agriculture, the primary safeguard of every nation against want. The danger of disease and pest introduction is real. For example, many hundreds of diseases are known to occur in other parts of the world which, like the Andean buba disease of potato, the Japanese brown rot of apple and pear, and a number of virulent grain rust races, have not yet reached North America; and everywhere nature is continually creating new strains of pathogens. Furthermore, disturbed world commerce coupled with expanded volume and speed of air traffic have tremendously increased the chances for disease introduction. Intelligent husbanding of agricultural assets is vital to permanent national welfare and demands that in providing defense against foreign pests special emphasis be given to those crops on which the country depends most. Since many phases of national interest are involved and quarantine funds and facilities are limited, some logical way is needed to establish a priority for crops which deserve special protective effort. A method for estimating crop importance has been suggested to me by W. A. McCubbin. He proposes a rating for each crop based on a summation of values assigned in each of several graded factors, namely crop use (food, feed, manufacture, soil-building, condiment, etc.); range of culture; quantity produced or money value; availability of domestic substitutes; and availability of foreign supplies. An estimate of the national importance of a crop thus obtained would rest on a broad base of facts, but other important factors cannot be disregarded in determining practical quarantine priorities. Each crop must be considered from the standpoint of the nature of its foreign pests and the adequacy of information about them, the relative cost and effectiveness of applicable quarantine measures, the effects of such measures on trade, the interests of producers and dealers, and the extent of public support. Decision as to the quarantine course to be recommended for the protection of any crop should rest primarily on a long-range view of the ultimate value of such a course to the country's agriculture. The decision will be modified, however, by the impact of such limiting factors as those just mentioned. Moreover, emphasis on the protection of major crops should not bar the protection of minor crops and other plant life where this appears feasible and in the national interest. The problem of adequate plant quarantine protection calls for the fullest utilization of existing knowledge and the aid of intensified research to provide a basis for intelligent action. Information is needed not only on foreign pathogens as such—their geographic and host ranges, race variations, life habits, and ecological relations—but also on their possible means of entry, their ability to attack domestic varieties, and their amenablity to treatment. In this field plant pathology has an indispensable service to render and its participation becomes more and more important as the destructive potentialities of foreign plant disease introduction and the value of defense against it become more generally recognized throughout the world.

Emphasis on the Study of Foreign Diseases. N. REX HUNT. Emphasis on the study of foreign diseases must be increased, owing to their probable wartime spread and the increased risk of introducing viable pathogens by fast air freight, if we are to meet our needs for information in promulgating and modifying quarantines; in finding diseases on material offered for entry or as soon as possible thereafter; in treating incoming material to eliminate risk of introduction of new diseases; in evolving eradication methods for diseases gaining temporary establishment in spite of quarantine vigilance; in providing treatments for control when eradication is impractical; in determining in advance of establishment of a foreign disease what the susceptibility of our commercial varieties and breeding stocks may be, so desirable resistant varieties may be supplied as promptly as possible when needed; in supplying data in advance of possible entry of a destructive disease of a major crop so agricultural planners may be prepared to maintain adequate supplies from other sources and to provide substitute crops for stricken areas. As aids in studying foreign disease problems we have indexes to the literature; the 600,000 records of diseases and insects found on incoming plant material are cross indexed by pathogen or pest, by host, and by country of origin and give a good idea of the types of pathogens and pests transported in commerce; and annotated lists of foreign diseases of important hosts are being prepared to supplement Stevenson's Manual of Foreign Plant Diseases which is now out of date. Plans for postwar Agriculture are being discussed. Provision for all needed work on plant diseases should be an integral and coordinated part of the plans. With respect to the study of foreign diseases, consideration should be given to the following: (1) The possibility of cooperative preparation and issuance in readily usable form of information already available regarding foreign diseases, and of keeping such information current. (2) Development of better coordination and wider use of correspondence and cooperative arrangements with foreign pathologists, with State Department representatives and with others abroad; of more effective arrangements for obtaining conferences with visiting pathologists from abroad; development of a general plan for stationing pathologists abroad temporarily, especially for the study of destructive diseases which cannot be studied here with safety and cannot be adequately studied through literature and by correspondence, in order that we may have these pathologists available for consultation here when needed; development of a comprehensive plan, in connection with the foregoing arrangements, for testing the reaction of American varieties and breeding stocks to foreign diseases. (3) Development of techniques to enable quarantine inspectors to detect foreign diseases, and of treatments to render incoming material safe for entry. At present most bacterial and virus diseases and thousands of fungous diseases in imported material are not determined, partly due to lack of techniques, partly due to lack of pure culture facilities; and for many foreign diseases no safe treatment is known to us. (4) Materialization of the plans made will require wholehearted public support based on a realization of the need for the work. Hence provision should be made for maintaining a constant and effective flow of information regarding the menace of foreign diseases, appropriate material to be supplied to agricultural, horticultural and gardening publications, to newspapers and magazines, to teachers in schools and colleges, to extension workers and any others needing it or able to use it effectively.

The Problem of Excluding Foreign Virus Disease. LEE M. HUTCHINS. Importation of living plants and parts from foreign countries is so intimately associated with improvement in agriculture, horticulture, and other plant industries, that it cannot conceivably be discontinued to alleviate the hazard of introducing dangerous plant diseases. The problem before us therefore is to determine what practicable measures will provide the greatest protection against the entrance and dissemination of these diseases in view of a continuing commerce in plant commodities. Exclusion of foreign virus diseases presents one of the most baffling aspects of this whole problem, complicated as it is by rapid airways transportation, fresh material shipped under refrigeration, symptomless carriers, and difficulties of detection in bulbs, tubers, rootstocks, cuttings, and dormant plants. Quarantine agencies need our full cooperation. Safeguards to be considered include: (1) knowledge of virus diseases occurring in foreign countries, to be obtained through literature, correspondence, travel and attendance at meetings, (2) appraisal of inspection, control, and quarantine methods as practiced abroad, and of relative hazards through plant shipments from the different countries, (3) stationing one or more of our pathologists at strategic locations abroad, to become familiar with virus diseases and keep us informed on current developments, (4) by cooperation with foreign governments and experiment stations, growing in foreign countries native American species of valuable trees and other plants and thus determining the diseases to which they are susceptible; in this way finding virus diseases dangerous to our species, before they reach our shores, (5) fumigation to free imported material of possible insect vectors, (6) evaluation of heat treatments and other methods of inactivating viruses in imported material, (7) detention and observation of questionable material under controlled growing conditions in

the United States before release, (8) prohibiting entry of certain material, where hazard of introducing a dangerous virus is known to be great, (9) through federal and state cooperation, inspection of imported plants where they are being grown in the United States, (10) inoculating the same or related species in America with inoculum from imported material, where this is warranted, (11) organizing virus symposia at national and international meetings of pathologists, and disseminating useful information on foreign virus diseases. An enormous reduction in importation of living plants, occasioned by world war, furnishes an unusual opportunity to improve protective measures against foreign diseases, for application at the cessation of hostilities and the resumption of normal commerce.

The Possibilities of Detention Procedures in the National Quarantine Scheme. R. KENT BEATTIE. Imported plants now spend but little time in transit to America. In the days of the sailing ship, weeks ensued, but the airplane has brought us to within 60 hours of the farthest parts of the world. The danger of the introduction of plant disease organisms in a living condition on imported plant material has correspondingly increased. In many countries, the plant diseases are entirely unknown. Commerce with these is rapidly increasing. Foreign inspection and certification, inspection, fumigation, and treatment of plant material at the port of entry are beneficial but only partially meet the danger. The port of first arrival for an airplane may be far within the interior of the United States. Many disease-producing agents are concealed in the tissues of the host with no external symptoms at the time of importation. They are out of reach of inspection and treatment. Especially is this true of such diseases as the Dutch elm disease, persimmon wilt, and some virus diseases when carried within the tissue of a resistant host. When a new disease enters it may destroy an established American crop or forest tree, or if controlled it may add greatly to the difficulty or expense of growing the crop or tree. Safety can be obtained only by growing introduced plants under control and observation until danger is past. This means reducing the quantity imported to the absolutely essential material and supplying adequate facilities for growing the imported plants isolated from American crop areas. Such isolation is already an established procedure in the control of animal and human diseases.

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